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SOME POINTS BEARING ON THE ASSOCIATION OF SENSORY DISORDERS AND VISCERAL DISEASE.

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In the following pages I deal in a somewhat discursive and desultory manner with the result of several years' enquiry into the symptoms evoked in the sensory nervous system by disease of the viscera. For many years the subject has been present in my mind, and although for a long time I could not appreciate the value of all the phenomena, yet by carefully noticing each circumstance in every case, and by grouping the symptoms together, I formulated for myself certain tentative laws of a somewhat vague nature, trusting that with extended experience these would become more defined and be of a less general nature. What I have published on this subject[1] does not fully accord with the results obtained by Dr. Head,[2] and although in many respects his results are more reliable than mine, there are a few points on which we are in conflict. These points may seem to be here unduly emphasised, but my object in stating them is that a subject of such importance and of such practical utility should be established on a basis

[1] *Medical Chronicle*, August, 1892.

[2] BRAIN, Part I. 1893.

which cannot be gainsayed, and that the views of Dr. Head will be enhanced if he is able to rebut objections. On the other hand, these views may be all the better for being modified if they are found irreconcilable with fact. While I, therefore, take exception to some of his conclusions as being too exact, yet I heartily congratulate him on a brilliant and valuable piece of work, and only regret that space does not permit me to emphasise the many points in which my evidence fully supports his conclusions.

In pursuing the study of this subject it is necessary to divest the mind of all preconceived ideas bearing upon it and to note carefully every individual circumstance, however contrary it may appear to one's expectations and desires. One great difficulty is the unreliability of the patient. Pain of the most excruciating kind leaves in the mind but a very imperfect recollection of its exact site. In all cases the description of the pain prior to examination should be received with great caution, and instructions given to note accurately the exact site during an exacerbation or recurrence. In the latter case trustworthy accounts can generally be received even when the observer is not present.

I must confess I have also found it necessary to put a control on my own observations, by having them verified by a colleague. In making my observations I invariably had the most striking and important cases examined independently. Such phenomena as my colleagues could not verify I rejected, for if these sensory symptoms are to be of value they must be capable of being elicited by observers free from bias. And further, cases should not be recorded based upon recollections alone, for it is wonderful how far from the truth the memory may wander, when there is a favourite theory to be advanced.

I.—The Pain in Visceral Disease is a referred Pain.

When I had for some time, in systematically examining patients, employed the site of pain as an important aid to diagnosis, I gradually became impressed with the apparent fact that there was little or no evidence pointing to the situation of the pain in disease of the viscera, being situated

in the organ affected. This view was strengthened by the results of *post-mortem* examinations in two cases of gastric ulcer, where the sites of the ulcers bore no relation whatever to the seat of pain during life. When Ross[1] published his highly suggestive paper, I was greatly helped in obtaining an explanation of many of my observations. In conversation with Dr. Ross I dissented from his view of the so-called splanchnic pain, maintaining that there was no evidence whatever in support of it. He stoutly maintained the contrary opinion, and used as an illustration a case of gastric ulcer that he had then under observation, in whom there was a pain of great severity localised in a limited area. He said he felt convinced that, were he to push a long needle into this painful spot, the needle would inevitably pierce the ulcer. In opposition to this view (which is very widely held), I urged the fact that whilst the site of pain was absolutely stationary, the stomach (and its ulcer) executed movements of a considerable excursion, both by reason of the musculature of the stomach itself, as well as by the mechanical movements of respiration.

My attention being somewhat accidentally called to the presence of hyperæsthesia in a patient recovering from an attack of gall-stone colic, I made careful search for this phenomenon in other cases, and collected a large number of illustrative cases, intending in the first instance to submit them to Dr. Ross as evidence in favour of the somatic origin of his splanchnic pain. But this was never done, on account of his painful illness and deeply regretted death.

The sensory phenomena, besides pain, when present, exhibit an increased irritability of the peripheral nerves. This is evidenced more particularly by cutaneous hyperæsthesia, and exalted sensibility of certain sub-cutaneous tissues of the external body wall. The first of these can be demonstrated by the usual methods for testing the hyperæsthesia of the skin, while the latter can be elicited by lightly grasping the various structures through the skin between the thumb and fore finger. There can occasionally be demonstrated a differ-

[1] Ross, J., "On the Segmental Distribution of Sensory Disorders," BRAIN, January, 1888.

ence in the sensibility to this stimulation in the various sub-cutaneous tissues. Thus, when no hyperæsthesia of the skin exists, as tested by lightly stroking with a blunt pointed instrument, when the skin is grasped in the above manner, abnormal tenderness may be elicited. Similarly when the skin is not unnaturally sensitive the sub-cutaneous fat or mammary glands may be so. And again, while these structures may exhibit no abnormality, certain muscles may be extremely hypersensitive. Further, distinct tenderness may be found in pressure over certain vertebræ, and that when there is no increased sensitiveness of the tissues interposed between the vertebræ and pressing thumb. These tender vertebræ frequently afford very valuable evidence of the organ diseased. When hyperæsthesia of the skin is present along with this tenderness of the sub-cutaneous tissues, the two areas are not co-extensive—the tenderness of the sub-cutaneous tissues generally being spread over a larger area than, and even manifested at some distance from, the cutaneous hyperæsthesia. In such cases, if the nerve supply of the organ affected be carefully enquired into, an explanation will be generally found in the fact that the nerves supplying the organ are in connexion with the spinal nerves at different levels of the cord.

Pain, when felt by the patient in whom there is also increased sensitiveness of certain areas of the skin or sub-cutaneous tissues (limiting the term sub-cutaneous tissues to the structures of the external body wall), is generally situated within the area of hypersensitiveness. Now as these areas are these to which Ross usually referred his splanchnic pain (the pain in the organ itself), it seems but reasonable to expect that the pain would be produced in the nerves thus abnormally excited. That is to say, if the roots of certain nerves are excited to a degree that produces this increased sensitiveness of the external body wall, it may be reasonably assumed that the irritation of the nerve roots also produces the pain which is felt in the area of their peripheral distribution. That a pain can thus be referred has been accepted by many writers, and Ross adopts it in the explanation of the origin of his somatic pain. The only difference between this referred pain—the somatic—and the so-called pain of the organ

itself—the splanchnic—is one of locality, the latter having its seat in close relation to the situation of the organ affected. If we take the pain of renal calculus as an illustration, it will be found frequently that the pain extends from the lumbar region round the side and down the abdominal wall slanting inward to the scrotum with, it may be, retraction of the testicle. As it is known that the ureter occupies a somewhat similar situation, it is frequently assumed that the pain in such a case is felt in the ureter itself. But the 12th dorsal nerve in its peripheral distribution follows a somewhat similar course, and frequently after an attack of renal colic, the skin here is hyperæsthetic. It seems, therefore, much more reasonable to assume that the pain here is in reality due to some irritation at the root of the 12th dorsal nerve, and that the pain is felt in the area of cutaneous distribution in accordance with the law of eccentric projection of pain. This assumption is all the more reasonable when there is evidence of the irritation having extended to more distant nerve roots, as when the pain and hyperæsthesia extend to the regions of distribution of the 1st and 2nd lumbar nerves, and even having affected the motor centres, as evidenced by the retraction of the testicle, and feeling of loss of muscular power in the muscles supplied by those nerves.

But it is in the study of the pain in heart affections that the most striking evidence is furnished in favour of the view that the pain in visceral disease is not located in the organ. Many precise observations have been made supposed to demonstrate the sensitiveness of the organ. Walshe[1] and Balfour[2] speak of the heart becoming painful, and Peter[3] has assumed that he could demonstrate this sensitiveness by exerting pressure on the organ. But in such cases where the exertion of this pressure elicits pain it can be readily demonstrated that the pain is really elicited from stimulation of the external tissues.

The hyperæsthesia of the skin associated with attacks of

[1] Walshe, W. H., "A Practical Treatise on the Diseases of the Heart and Great Vessels," London, 1873, 4th edition, p. 208.

[2] Balfour, G. W., "Clinical Lectures on Disease of the Heart and Aorta," London, 1876, p. 274.

[3] Peter, M., "Maladies du Cœur," Paris, 1883, p. 36.

angina pectoris is no new thing, several writers mentioning the observation. It is needless to affirm, however, that a great many cases of angina pectoris suffer typical attacks without any symptom of peripheral hyperæsthesia, either of the skin or subjacent tissues. Such a case presented itself to me a short time ago where the patient died in the second attack of pain. In him no other sensory abnormality whatever could be elicited. The pain was situated across the lower third of the chest, extending slightly to the left. In such a case one might assume that the pain was undoubtedly in the heart. But is it so in reality? During the period that a patient suffers from an attack of angina pectoris the heart's movements still continue. The movement of such an abnormally sensitive organ would surely cause some variations either in the intensity or in the situation of the pain, and of this there is no evidence. Further, the pain of angina pectoris very often extends over a greater area than that covering the heart. In many cases this extension of the area takes place in a manner very instructive. In the first instance the attacks of pain may be limited to the præcordial region. As they increase in severity they extend further round the left chest and towards the arm-pit, and it may be down the inside of the left arm. The pain appears to be exactly of the same nature in the more distal parts as it is when it is located in the præcordia. It is a mere assumption to say that in the one instance the pain is in the heart, and when felt in more distant regions that it is due to reflex irritation of the spinal roots, particularly when the cutaneous area over which the pain is felt is supplied by nerves whose spinal roots are in immediate contiguity. This is more strikingly demonstrated when hyperæsthesia of the skin is likewise present. In such a case the pain is situated within the hyperæsthetic area, and this area can be demonstrated to be the region of cutaneous distribution of the 4th, 3rd, and 2nd dorsal nerves. The pain may even not be felt in the region of the heart at all. From the study of a series of cases extending over a period of years during which I have observed the attacks of angina pectoris first appear and develop, I have been able to observe a great diversity in the modes of attack. Although the symptoms of

pain are usually præcordial at first they are not invariably so. In one patient whom I have had under observation for two years with symptoms of aortic stenosis, the pain and hyperæsthesia in the first instance were situated over the anterior axillary wall and in the arm-pit. Lately the attacks have developed with extreme severity, and the hyperæsthesia now embraces the upper part of the left chest with the inner half of the left upper arm, the ulnar half of the forearm, and the little finger. There is also extreme sensitiveness of the left sterno-mastoid and trapezius muscles. This distribution of the pain and hyperæsthesia is so characteristic, as to leave no doubt of the distinct connection of the cardiac nerve supply with the nerve roots in the spinal cord supplying those hypersensitive structures. Thus the hyperæsthesia of the chest and arm is practically the field of cutaneous distribution of the upper four dorsal nerves of the left side, the roots of these nerves being connected with the sympathetic fibres that supply the heart. The tenderness of the sterno-mastoid and trapezius muscles, indicate irritation at the root of the spinal accessory nerve, and it apparently receives the stimulation from the heart through the medium of the vagus, the roots of the vagus and spinal accessory being connected. The sensory phenomena exhibit great variation in the locality of this distribution. Sometimes the dorsal nerves show no cutaneous hyperæsthesia, while the cervical nerves do. I have at present under observation a patient who has marked attacks of angina pectoris, and these I have noted developing during a number of years. In her there is very slight tenderness of the deeper tissues of the thoracic wall, whilst the left sterno-mastoid and trapezius muscles are extremely tender and the hyperæsthesia of the skin is limited to the left side of the neck, in an area corresponding to what I take to be the peripheral distribution of the 3rd cervical nerve.

But while most of the cases show the pain limited to, or originating in the præcordial area, other rare cases, manifest attacks of angina pectoris, which have no relation to the chest wall. In such cases the term angina *pectoris* is certainly a misnomer, but taking the term to imply that severe pain associated with disease of the heart, it will be found to

appear typically in regions distant from the heart. The following case is such a striking illustration of this, that on account of its importance I quote it at length.

Case of Chronic Fibrous Myocarditis and Calcification of the Coronary Arteries, with severe pain in the left forearm.—Female, age 60, consulted me on 30th July, 1892, complaining of great irritation in the privates which comes on periodically and sets up a great pain that strikes into the left forearm. These attacks occur several times a day, and last from fifteen to twenty minutes. She feels great depression when the attack comes on. She is very short of breath, and there is slight swelling of the legs. There is considerable difficulty in making water. There is great thirst, fair digestion, and constipated bowels. The patient is pale and wan, and the face has a pained and anxious expression. The body is well nourished. The tongue is very dry. The urine has a specific gravity of 1030, contains a trace of sugar, but from enquiry the quantity of urine passed does not appear to be abundant. The site of the irritation is very definitely limited to the inner surface and skin edge of the vulva. The arteries are large and slightly atheromatous. The pulse is full, soft, quick, and regular. The left side of the chest is tender to pressure, but there is no cutaneous hyperæsthesia. There is a very tender spot on pressure over the 2nd left rib in the nipple line, and the upper 3rd dorsal vertebra is very tender. There is no defined apex beat. The area of the heart's dulness begins at the 3rd rib in the parasternal line, and extends transversely from the middle of the sternum outward to the left for four and a half inches. The heart sounds are sharp and clear in all the areas, but in the aortic area the second sound is markedly accentuated, resembling a drum-tap. There is marked pulsation of the internal jugular veins, and tracings of this pulsation taken at the same time as the radial pulse, show the pulsation to be due to the contraction of the right auricle.

No abnormality can be detected in any other organ. The patient was directed to diet herself carefully, and, for relief of the irritation, to inject hot water into the vagina by means of a Higginson's syringe during the attacks and systematically night and morning. She was asked also particularly to note the exact site of the pain when it recurred and its connection with the irritation in the vulva.

In ten days' time she returned. The pain she found had no relation with the irritation, but came on always with exertion. It was situated in the left forearm, and she defined very clearly the

ulnar half of the forearm. Sometimes it was associated with a pain of less severity under the left breast. Under the treatment prescribed the irritation was greatly relieved, and the attacks had become much rarer.

The progress of the case was satisfactory so far as the urinary condition was concerned. The sugar after a few weeks' treatment disappeared from the urine, and the irritation also completely subsided. But the pain in the arm resisted all treatment. On one occasion she came to my consulting room in a terrible state of distress, and sat rocking the body to and fro, nursing her left forearm in her right hand. The onset of this paroxysm of pain had been induced in consequence of her having to hurry to catch the train by which she was coming to consult me. While she was under this attack I made a careful observation of the pulse and the site of pain. The pulse presented no change from its usual character, either to the touch or from the study of the sphygmogram. *The pain was exactly located in the ulnar half of the forearm*, no pain being complained of in any other part.

As I could give the patient no relief, she came into the Victoria Hospital under my care. She was put to bed, and not permitted to get up for three weeks. After the first week she had no attack, and after she was discharged she had them only very slightly and very rarely. I lost sight of her for a few months until the 9th of July, 1893, when she consulted me for an attack of diarrhœa. She said the old attacks of pain had never been so frequent since she had left the hospital, although occasionally they were still very severe, but were always limited to the left forearm. On examination during this visit I found only slight tenderness of the 1st dorsal vertebra and of the sub-cutaneous tissues in its neighbourhood. The heart presented no change except that the second sound, although still markedly accentuated, was not quite so clear, being somewhat muffled. She had her left forearm carefully wrapped in flannel, under the impression that it eased the attacks. There was no sensory abnormality demonstrable in the arm. On the 22nd I was asked to see her, as she was very ill. I saw her on the 24th. She was propped up in bed suffering from great dyspnœa, and the legs were greatly swollen. She died next morning.

Permission to examine the heart was obtained with difficulty, and only an incomplete *post-mortem* was therefore held. I sent the heart (which was empty and flabby) to Dr. Williamson, of Manchester, and his report is as follows:—

Heart weight, 10½ oz. Left ventricle slightly enlarged, very slight thickening of aortic valves. Pulmonary, mitral and tricuspid valves normal. No atheroma of the aorta. The muscle wall of the left ventricle is of normal thickness and consistency; but in the cut surface there are a number of small pale whitish patches of irregular size and shape. Both coronary arteries are markedly atheromatous and calcareous from their aortic origin down to their small sub-divisions. The main arteries and their chief branches can be dissected out as calcareous tubes. The cardiac muscle of the left ventricle when examined microscopically does *not* present any signs of fatty degeneration (osmic acid). Sections show patches of fibrous tissue amongst the muscle fibres.

The pathological diagnosis is *marked atheroma and calcification of the coronary arteries; chronic myocarditis (fibrous).*

There can be little doubt that the cause of the pain in this case was situated in the heart. Putting aside the situation of the pain, all the other phenomena pointed to the heart as being the *origo mali;* as, for instance, the suddenness of the attack engendered by exertion, the dull, terrible agonising nature of the pain, and the great sense of oppression accompanying it. The peculiar face of the patient was to my mind also characteristic of the expression in patients who suffer much pain associated with disease of the heart. Concerning the situation of the pain, it was at last entirely situated in the arm. Occasionally at first she did complain of pain under the left breast, but in the few attacks which I have witnessed the pain was entirely located in the arm. And the area here was extremely definite in its limitations. When asked as to its situation she always stroked the ulnar border of the forearm from the elbow to the wrist. There was never any other disorder of sensation in this locality, neither of the skin nor deeper tissues.

The area, then, in which this pain was felt was in the region of cutaneous distribution of the 1st dorsal nerve. Considering the marked disease of the heart substance and of the coronary arteries, there seems little doubt but that the stimulation from them proceeded to the root of the 1st

dorsal nerve, and the pain was then referred to its cutaneous distribution. Suppose now that the area of pain had corresponded to the area of distribution of the 3rd or 4th dorsal nerves in the chest—a circumstance far from unusual—would not one have said then that the pain was felt in the heart itself? And this view would have seemed all the more probable if there were complete absence of hyperæsthesia. It seems to me, in view of such a striking example as this case presents, that there is room for great doubt that the pain of an organ is necessarily situated in the organ simply because the pain may happen to be located in its neighbourhood. The pain in angina pectoris may extend in a direction the reverse of the course usually pursued, originating say at the wrist, and extending up the inside of the arm to the chest. Here it would seem that the irritation first affecting the root of the 1st dorsal spreads to neighbouring roots—characteristic of the manner in which the area of pain extends in diseases of other organs. Yet Walshe[1] looks upon such a manifestation as being an instance where "the brachial pain played the part of an aura."

Studying the subject from a purely clinical aspect, such are, in brief, some of the reasons that influence me in considering the pain of visceral disease to be of a referred character, always somatic, never splanchnic. I willingly admit I am not able to explain the source of all pains, and many patients complain of pain to me unexplainable. Yet with this view present in my mind, and with extended experience these cases become less numerous.

II.—The Sensibility of the Visceral Tissues.

A certain amount of light is thrown upon this subject when the sensitiveness to stimulation of the viscera is considered. The relative great bulk of the organs to the nerves that supply them and the varied functions these nerves subserve, suggest the idea that there can be but a very limited amount of sensibility.

[1] *Loc. cit.*, p. 198.

In considering this subject it will be convenient to divide the tissues constituting the viscera into three, namely, the serous, muscular and glandular. This division will be useful in considering not only the sensitiveness of the tissues to ordinary stimulation, but also in considering the sensory disorders that arise during affections of the individual organs. To the methods employed for ordinary stimulation (touching, pricking, pinching, &c.) it is well known that the viscera are insensitive. But still, painful sensations can be aroused, and the symptoms thus elicited help in clearing up a good many obscure phenomena.

(A) *Serous Membranes.*

For many years when tapping for pleurisy I have been accustomed to note the sensations elicited when the visceral pleura came in contact with the canula. Coughing has been set up, but never any pain or other sensory phenomena. In like manner on testing the parietal and visceral pleuras with more facility in cases of excision of the ribs, I have failed to elicit any painful sensation. Richerand[1] long ago noted the insensibility of the pericardium, which he exposed in removing a portion of a scirrhus pleura. The insensibility of the peritoneum has been frequently demonstrated in operations affecting the various abdominal and pelvic viscera.

(B) *Muscular Tissue.*

In hollow muscular organs pain can be elicited by stimulating the muscle to contract, as after giving a distending enema the great gut contracts producing a referred pain in the abdominal wall midway between the umbilicus and symphysis pubis. The uterus can be made to contract, with the production of the pain in a similar area. The pains may extend in both cases to other regions, but in no sense are they felt in the contracting organ. But to any stimulation, save that which gives rise to powerful contraction, the muscular is as insensitive as any of the other tissues.

[1] Richerand A., "Elements of Physiology." Edited by James Copland. 2nd edition, 1829, p. 613.

The insensitiveness is still marked when the parts are inflamed. In a patient from whom a large piece of gangrenous intestine had been removed, the exposed gut a few days after was deeply congested and swollen. But stimulation of the peritoneal and mucous surfaces and intervening tissues was not perceived by the patient.

Seeing that it is the muscular tissues that evolve pain most readily, it will be best to study shortly the sensory phenomena associated with the hollow muscular viscera. No one doubts but that the pain felt during painful peristalsis of the bowel, or during uterine contractions, is produced by the muscular tissue. The pain so produced may be still felt after the contraction has ceased as a feeling of soreness, and hyper-æsthesia can frequently be demonstrated. It is a curious fact that the pains, and other sensory disorders that are most severely felt by patients, are those that arise from affections of the hollow muscular organs. I do not mean to imply that spasm of the muscle is necessary to the production of pain. In angina pectoris, this is manifestly not the case, notwithstanding many opinions to the contrary. But the pains, for instance, that are the most marked and can be referred with most certainty to the organ affected, are those of the heart, of the stomach, small and great intestine, and of the uterus. The pains, likewise, of gall stone and renal calculus are in all likelihood essentially those of hollow muscular organs. The calculus lodged in the substance of liver and kidney gives rise to no sensory symptom, but when it invades the hollow viscera connected with them then the pain is aroused. I have had frequent occasion to observe the persistence of pain, and hyperæsthesia after the expulsion outside the body of gall stone and renal calculus. In view of the possibility of these symptoms, and those also manifested so frequently in stomach affections, being associated with and probably due to spasm of muscular tissue, the following remark by Beaumont in his observation upon Alexis St. Martin is of interest.[1] "If the bulb of the thermometer be suffered to be drawn down to the pyloric extremity, and de-

[1] Beaumont, W., "Experiments and Observations on the Gastric Juice, &c." Edited by A. Combe, Edinburgh, 1838, p. 105.

tained there for a short time, or if the experiment be repeated too frequently, it causes severe distress, and a sensation like cramp or spasm, which ceases on withdrawing the tube ; but leaves a sense of soreness and tenderness at the pit of the stomach." I have little doubt in my own mind, but that the tenderness and soreness here described, was in reality due to a condition of hyperæsthesia of the skin or subjacent tissues. I usually find these phenomena when patients complain of such feelings in the epigastrium. In a case of lax abdominal walls Macdonnell[1] was able to palpate the under surface of the heart, and pressure upon the auricles gave rise to symptoms resembling angina pectoris. Without seeking, therefore, to insist that the pain in visceral disease is due in all cases to muscular spasm, I would seek to point out, first, that pain and other sensory phenomena are frequently associated with hollow muscular organs ; second, that symptoms exactly analogous to these produced by disease can be originated by artificial stimulation of the muscles of hollow organs.

(c) *Parenchymatous Tissues.*

Diseases of the parenchyma of an organ are not associated with very definite sensory symptoms. In fact, I have more than a suspicion that there are none. I have carefully enquired into the history, and watched the progress of many cases of acute nephritis from the onset, and yet have been unable to find any sensory symptom associated therewith. The ravages of consumption are notoriously painless, unless the pleura be involved. The pains of pneumonia are those observed in pleurisy.

A certain amount of pain can be elicited by pressure on a number of the organs (kidney, testicle, ovary, &c.). But here, so far as I have been able to make out, the pain is a referred pain.

When pain occurs associated with disease of an organ, it can frequently be demonstrated that there is an extension to the serous covering, as in the pleurisy of pneumonias. I have had frequent opportunities lately of studying the pains associa-

[1] Macdonnell, H., "Cardiograms from the Human Heart," *The Practitioner*, vol. xliv., 1890, p. 178.

ted with great enlargement of the liver from disease of the heart. In such cases pulsation of the organ is a frequent symptom, and in the course of an enquiry into the nature of this pulsation, I noted the frequent occurrence of pain over the distended organ. So far as I could make out it was evidently on the surface of the organ, and in one case where the pain and tenderness were very marked, there were found in the *post-mortem* examination numerous adhesions between the anterior surface of the liver and the abdominal wall.

I have already referred to the pains associated evidently with the ducts of certain organs (bile ducts, ureters), and it is a fact worth noting that diseases of a ductless gland like the spleen appears to be unaccompanied by any sensory symptoms. One great obstacle in regard to this theory of the painlessness of parenchymatous disease, is the pain associated with orchitis, unless it be due to the swelling pressing on the more sensitive tissues external to the organ. Otherwise, why inflammation of this organ should produce such pain, and inflammation of the kidney should be painless, I do not understand. I am conscious that many very capable physicians affirm with great assurance that there are pains distinctly associated with disease of the parenchyma of organs. But all I can say is that looking at the matter dispassionately I have yet found no distinct evidence of such a thing, and I do not feel justified in ascribing a pain, whose origin I do not know, to an obscure organ whose sensory manifestations I do not understand.

III.—The Character and Distribution of the Sensory Symptoms in Inflammation of Serous Membranes.

When all the sensory symptoms that can be elicited from a series of cases of inflammation of the pericardium or pleura, are grouped together, it seems very difficult to find a satisfactory explanation for them.

The assumption that the pain is local and due to stimulation of the peripheral nerves, the view most commonly held and supported by Ross and Head, as distinct from a reflex or associated pain, is not sufficient. A great variety of these sensory phenomena may present themselves in cases that are otherwise apparently identical so far as our means of physical

diagnosis are concerned. In looking over my notes of six cases of pericarditis I found that two presented no sensory phenomena, that a third had a slight pain referred to the upper part of the præcordial region, that a fourth had pain for a few hours of agonising severity across the middle of the sternum. I was summoned to see this patient between three and four in the morning, and the symptoms resembled those of an attack of angina pectoris—the ashen hue of the patient's face, the fear of impending death, and the agonising pain. The pulse was quiet and regular, and the only abnormality was a to and fro murmur over the area of the heart's dulness. After the subcutaneous injection of a large dose of morphia the pain abated. Later in the morning the pain had entirely gone, and the friction murmur had nearly disappeared, and in a couple of days had entirely disappeared. Since then (three years ago) the patient has had no recurrence. The last two cases presented somewhat similar phenomena; one of them only I will detail.

Case of Pericarditis with marked sensory disorders.—Male, aged 23, thin and meagre, had been under my care occasionally for nine months, chiefly on account of obscure abdominal symptoms (pain and diarrhœa). I had kept him under observation chiefly on account of marked pulsation in the veins of the neck and sometimes in those of the chest wall. The venous pulse was of the auricular type, *i.e.*, the main wave of the pulse was due to the auricular systole. There was also a marked extension of the heart's dulness to the right of the middle line for two inches. He had occasional attacks of violent palpitation in the night. On the 18th March, 1893, he left his work at eleven in the morning, feeling tired and exhausted. On the 21st he was seized with a pain of great severity in the chest, with difficulty in breathing. I saw him on that day and found him sitting up in bed, holding his body very rigid and breathing with evident difficulty, and on his face an expression indicative of great suffering. The pain struck from the præcordial region round to the left shoulder blade (so that he could not lift his left arm) and down the inside of the left arm and ulnar part of the forearm to the little finger. Any movement of the body caused pain, and he was too terrified to take a deep breath. There was extreme tenderness of the skin and sub-cutaneous tissues over the whole front of the chest as far out on both sides as the nipple line. Over the 3rd left costal

cartilage and adjacent portion of the rib, this tenderness to pressure was excessively acute. Careful note was made that the tenderness referred entirely to the tissues in the external body wall, and that the method to elicit this tenderness did not affect the heart substance. The pectoral muscles, and the structures forming the anterior and posterior walls of both axilla were also very tender. There was also slight tenderness to pressure of the left sterno-mastoid and trapezius muscles, one point over the trapezius immediately above the middle of the scapular spine being especially tender. The muscles over the back of the scapula were also very tender. On auscultation over the heart, a loud to and fro friction murmur could be heard over the whole heart. There was no murmur accompanying the heart sounds. The pulse was quick, small and regular, 110 per minute. The temperature was 103° F.

For three days these symptoms remained practically unaltered, the pain only yielding to large doses of opium. During the time he was so ill I asked two of my colleagues to visit him, who verified the diagnosis and the above description of the symptoms.

(In view of the resemblance of the symptoms in these cases to those of angina pectoris associated with disease of the myocardium, the suggestion has occurred to me that it is possible that the pain, even of pericarditis, may be due to the invasion of the muscular tissue of the heart by the inflammation).

In pleurisy similar phenomena are sometimes met with. Thus in a case recently under observation, the onset of well-marked dry pleurisy gave rise to no pain whatever. The friction was at first limited to the right base, and attention was only directed to the condition by a sudden rise in temperature. The friction extended after a couple of days round to the front, and could be heard over the lower third of the chest wall; at the same time symptoms of pain and tenderness on pressure were developed. There was no cutaneous hyperæsthesia, and the tenderness was well-marked on percussing the chest. This tenderness extended higher than the friction sounds, and it is quite possible that the pleurisy extended over a greater area than that over which the friction was heard. There was no tenderness, however, of the chest wall behind the posterior axillary line, although the friction sounds were heard over this area in

the lower part of the chest. But the interesting point in this case was the tenderness of the muscles forming the anterior and posterior walls of the right axilla. Thus, on gently grasping the skin no increased sensation was produced, but on grasping as gently the muscles, then the patient winced from the severity of the pain. On the other hand, no symptom of pain was elicited on applying the same test on the left side. Without entering minutely into the particular nerves here manifesting this abnormal sensitiveness of the axillary walls, it may be pointed out that none of them came within the region of the inflamed pleura.

Hilton has remarked upon the sensitiveness of the skin in pleurisy, and evidently considers it as due to reflex stimulation of the nerve root, comparing it to the pain felt on the inner side of the knee, in disease of the hip joint. In fact, Hilton bases an elaborate argument upon the intimate association of the nerves supplying a joint, the muscles acting upon the joint and the skin covering the muscles, and looks upon the great serous cavities as being analogous to the joints of the extremities. I have frequently observed well-marked hyperæsthesia in cases of pleurisy, and that in a situation that could not be explained by the pleurisy affecting the branch of a nerve and so producing the hyperæsthesia.

Case of Basal Pleurisy with abdominal Hyperæsthesia.—Female, aged 31, complains of pain in her left side, especially on taking a deep breath. The patient is thin, but fairly nourished. She lies in bed slightly propped up. The temperature is 100° F. The respirations are quick (32 per minute) and shallow, and each respiration is suddenly checked. The pulse is 104 per minute, small and regular. There is great pain and soreness in the left hypochondrium. An area of hyperæsthesia can be readily mapped out, extending as a band from near the middle line in front round the left side of the abdomen to the spinal column. The breadth of the band is about three inches. In the front the umbilicus is situated opposite the middle of the band, and at the sides it crosses the tips of the floating ribs, and terminates behind opposite the 12th dorsal and 1st lumbar vertebræ. The edges of the band are not well defined. The chest is resonant on percussion, and on auscultation the only abnormality is a small

area over which friction is very distinct at the base of the left lung behind. These symptoms persisted for a couple of days, then disappeared together, that is to say, on the third day the temperature was normal, the respirations quiet and free from pain, the hyper-æsthesia was gone, and no friction could be detected.

I have, in a great number of cases of pneumonia, found a patch of hyperæsthesia over the area in the abdomen where pain is so frequently complained of in such patients. This abdominal pain of pneumonia is a well-known symptom, but its association with hyperæsthesia has not been sufficiently recognised, although Valsalva mentions it in a case quoted by Morgagni.[1] I have rarely found it quite so extensive as in the above case, and I have always viewed it as being associated with diaphragmatic pleurisy, a view that finds support in the symptoms of such cases as the one just recorded.

A shoulder pain associated with disease of the abdominal organs has long been recognised, and has been the object of particular study by Peter,[2] who associated it with the phrenic nerve, irritation being conveyed by this nerve to the roots of the cervical nerves, and the pain referred to their cutaneous distribution—a view that receives the support of Ross. In several cases of basal pneumonia and of pleurisy limited to the lower margin of the lungs I have found not only this shoulder pain, but also a distinct field of hyperæsthesia on the shoulder. In such cases I have attributed the shoulder pain and hyperæsthesia to the accompanying diaphragmatic pleurisy (although it may be due to the vagus terminations being involved by the inflammation of the lung). It is difficult to account for these phenomena satisfactorily. Hilton describes the parietal pleura as being supplied by intercostal nerves. If so, they must be very scant. Some years ago I made a careful dissection of several intercostal nerves, first following the trunk of the nerve and

[1] Morgagni, H., "De sedibus et causis morborum," book ii., letter xx., article 30.

[2] Peter M., "Névralgie diaphragmatique," &c. *Archives Générale de Médecine*, June 17, 1871.

tracing each twig to its destination, but I could not find a single filament going to the pleura.

If the suggestion be found tenable that the far-reaching pain and hyperæsthesia accompanying pericarditis is due to an extension of the inflammation to the myocardium, then the abdominal and shoulder pains of basal pleurisy may in like manner be due to an invasion of the muscular tissue of the diaphragm by the inflammation.

Of the sensory phenomena associated with disease of the peritoneum I have very little experience, and I am unable to give any definite results. I may say that since I recognised the hyperæsthesia associated with diseases of definite organs, I have had fewer cases of "peritonitis"—for sometimes the hyperæsthesia in, say, a case of pyelitis with retention of pus, gives rise to symptoms resembling the descriptions of extreme tenderness of the abdomen in cases of peritonitis.

When, however, the various sensory disorders associated with the diseases of the serous membranes are considered, there is some difficulty in getting a clear conception of how they are produced. At all events, the statement that "affections of the serous cavities of the body do not cause referred pain or cutaneous tenderness, but produce local pain which follows the lines of peripheral nerves, and is associated with deep tenderness over the affected point only" (Head) is not sufficient explanation for the conditions present in such cases as I have quoted above.

If this view of the absence of pain in the viscera be substantiated, it would lead one to surmise that the great organ of sensation, the skin, is altogether a later development in animal life, having for its function the protection of these viscera necessary to the maintenance of organic life. The sympathetic nerves would therefore have functions apart, and distinct from those of the spinal nerves. This independence of the sympathetic nervous system has received a very elaborate exposition by Davey.[1] Support

[1] Davey, J. G., "The Ganglionic Nervous System," London, 1858.

for this conjecture is found in the recent observations of Paterson,[1] who maintains that the sympathetic nervous system is developed independently in the mesoblast, and does not arise as an outgrowth from the cerebro-spinal system. The elaboration of this view necessarily leads to the discussion of matters pertaining to morphology and development—subjects which I am not qualified to discuss.

IV.—The relationship between the Areas of Cutaneous Hyperæsthesia associated with Visceral Disease, and the Areas of Cutaneous Distribution of Spinal Nerves.

In my earlier observations I had come to the conclusion that when there was a limited field of hyperæsthesia in any given case of visceral disease, it occupied a situation in some part of the cutaneous field of distribution of a spinal nerve, whose root was in intimate association with the root of the sympathetic nerves that supplied the affected organ. When the hyperæsthesia became more extensive, it spread in a somewhat diffuse manner into fields occupied by spinal nerves, whose roots are in close relation with the one originally affected. While, therefore, there was a continuity in the skin area affected, that continuity always occurred in a definite direction, namely, in accordance with the distribution of the spinal nerves whose roots are in close association. Thus, the hyperæsthesia in heart affections beginning in the 4th dorsal nerve distribution might spread up the chest, but instead of passing over the clavicle into the field of supply of the 4th cervical it passes down the arm along the fields of supply of the 3rd and 2nd dorsal. In spreading thus it did not necessarily affect the whole area of supply of one nerve root. Thus, one portion of the field of supply of the 4th dorsal nerve might be hyperæsthetic. As this area increased in size, it did not first spread over the whole field of supply of the 4th nerve, then invade the field of supply of the 3rd. Occasionally it might do so, but more frequently if the area was limited, as it usually is, to the anterior surface of the chest;

[1] Paterson, A. M., "Development of the Sympathetic Nervous System in Mammals," *Philosop. Trans.*, 1890.

this area would extend into the armpit and down the arm without involving the posterior surface of the chest. There does not appear to be any law regulating this spreading. Thus, in four cases of gastric ulcer at present under observation, in all of whom there has been lately vomiting of blood, an examination shortly after the hæmatemesis revealed the following conditions relating to the sensory symptoms. In two there are absolutely no sensory symptoms. In one there is well-marked hyperæsthesia in the epigastrium shading off into a less definite field of hyperæsthesia that extends from the level of the umbilicus round to the left to the spinal column, and up over the thorax and down the inside of the left arm, and the ulnar half of the forearm to the little finger. In the fourth case the hyperæsthesia occupies the front of the abdomen, reaching on both sides as low as the groin, with a tongue-like projection into the front of the thighs, similar to that Head maps out as being characteristic of the distribution of the 12th dorsal nerve. On the right the hyperæsthesia reaches as high as the umbilicus, and on the left as high as the ensiform cartilage. It does not extend backwards on either side beyond the anterior axillary line. In this latter case there is remarkable extension of the area in which the uppermost epigastric reflex can be produced. In many persons the reflex contractions of the rectus abdominis muscle above the epigastrium can be observed to occur in three divisions corresponding doubtless to the divisions produced by the tendinous intersections. In this patient stimulation of the skin as high as the nipple frequently (and sometimes even the skin in the axilla) will provoke a contraction of the upper portion of the rectus on the left side. The skin here is not hyperæsthetic in the slightest degree.

V.—Overlapping of Sensory Fields.

In very few of the cases could the field of hyperæsthesia be delimited with certainty. Parts at the border would be hyperæsthetic at one time, and when one would return there would be some doubt, or a distinct assertion that the part was not hyperæsthetic. In the centre of any limited area the hyperæsthesia was more acute. Under these circum-

stances I did not think that the hyperæsthesia associated with visceral disease afforded a sufficiently accurate clue to the delimitation of the areas supplied by individual nerves, and I looked upon this variation in the intensity of the hyperæsthesia as being due to an over-lapping in the distribution of neighbouring nerves. This view was strengthened by the fact that I had seen evidence of a slight extension of the hyperæsthesia into fields supplied by nerves whose roots were not in neighbouring association. I considered also that I had obtained evidence of this overlapping from the study of the eruption in cases of herpes zoster.[1] When Dr. Head published his extremely exact delimitation of the fields, I imagined that I had not been sufficiently accurate in delimiting my fields. I therefore made another series of careful observations, and although in a few cases I appeared to get results that partly supported his views, I must confess to having utterly failed in the great majority of cases. Even in the study of a few cases of herpes zoster I could not get them to fit into any one of his fields. To demonstrate these several points I will quote the following three cases that merit publication for other reasons.

Case of Herpes Zoster without eruption.—Male, aged 53, complains of pain on the top of the right shoulder extending down the arm to the thumb. The pain began about three weeks ago, and has continued ever since. Sometimes it comes on in severe paroxysms, accompanied by a disagreeable smarting sensation. He can scarcely bear his clothes to touch his shoulder, and he can't wear his braces over the right shoulder because of the pain it causes. The paroxysms of pain have prevented him sleeping of late, and in consequence he is getting tired and weak. The patient is a big, stout man of healthy appearance. When stripped there is nothing abnormal to be observed in the skin of the right shoulder or arm. On testing the sensation, the skin on the top of the shoulder is markedly hyperæsthetic. The hyperæsthesia occupies a very characteristic situation, which can be easily mapped out, as it is so acute the patient has no hesitation in noting when the stimulation passes from the healthy skin to the abnormally sensitive. It begins abruptly in the neck about the level of the 3rd cer-

[1] Mackenzie, J., "Herpes Zoster and the Limb Plexuses of Nerves," *Journal of Pathology and Bacteriology*, vol. i., p. 332.

vical vertebra, and extends round the neck at that level to the middle line in front. It extends from hence over the top of the shoulder down the outer surface of the arm, and radial half of the forearm to the thumb. The posterior border of this area extends from the 3rd to the 6th cervical vertebra, then slants outwards at the level of the spine of the scapula to the posterior surface of the shoulder, then down the back of the arm past the side of the olecranon into the forearm to the thumb. The anterior border descends in the middle line to the level of the clavicle where it slants slightly outwards to the level of the third rib. It then runs transversely out to the front of the shoulder, where it descends in the front of the arm and forearm to the thumb (fig. 1). This area, although so distinct, does not evince the hyperæsthesia with equal intensity in all parts. Thus in testing round the arm, when passing from the healthy skin, the hyperæsthesia at the borders is not

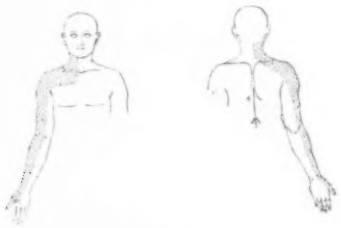


FIG. 1.

so acute as towards the centre of the affected area. The under surface of the thumb is slightly anæsthetic. The same results are obtained on testing by heat.

Careful enquiry was made into the condition of all the other organs, but with the exception of a slightly furred and fissured tongue no abnormality could be detected.

The patient called to see me at intervals of five or six days, and each time I mapped out the hyperæsthesia, the results always being practically the same. In a month from the date of his first visit the hyperæsthesia had gone, and with it to a great extent the pain. For several weeks later the pain used to recur in slight paroxysms.

I have assumed this case to be one of herpes zoster, although in the strictest sense it has no right to the term herpes or zoster. But that it is essentially the same disease I have little doubt. The description of the pain—the

paroxysms, its wearing character preventing sleep, and its duration are exactly the symptoms described by patients who suffer from herpes. Although I have never read of any account of herpes zoster without an eruption, I think that it may not be so very rare. Some years ago I saw in consultation a patient said to be suffering from some painful disease of the spine. In her there was a distinct area of hyperæsthesia following the distribution of the 4th dorsal nerve. The pains were characteristic, and located chiefly within the hyperæsthetic area, but radiating into the arm. I detected on the back two small vesicles, and on the strength of this, suggested the diagnosis of herpes zoster. The medical man in charge attributed the blisters to his applications, but on enquiry they were not such as would give rise to vesication. The future progress fully confirmed this view of the case. Lately I had a patient with severe pain in her side, and a distinct band of hyperæsthesia embracing one half of the body. I was a few days in doubt what might be the nature of the complaint until it was disclosed by the appearance of a few vesicles in the epigastrium, the remainder of the hyperæsthetic field remaining quite free. Dr. Head has likewise noted similar cases, and von Bärensprung,[1] has described the intervals of skin between the eruption as being hyperæsthetic. So that it would appear that an eruption is not a necessary symptom in herpes zoster.

On the question of differential diagnosis the only alternatives that occur to me, are that this case was an irritation of nerve roots due either to disease of the vertebræ or to some visceral lesion. That it was not due to a disease of the bones is evident from the progress of the case. There was never any spinal tenderness. I made, as I have said, enquiry into the condition of all the other organs in the head, throat, thorax, and abdomen, but could find no clue. The vagus and phrenic roots being in association with the cervical nerves led me to make careful examination of the organs supplied by them, but the quest was vain.

For these reasons I consider the case as being due to an

[1] Von Bärensprung "Die Gürtelkrankheit," *Annalen des Charité-Krankenhauses*, Band ix., Heft. 2, 1861, p. 103.

affection of the roots of the 3rd, 4th, 5th, and 6th cervical nerves, and to be of the same nature as that which gives rise to the disease known as herpes zoster. And the case illustrates in a very definite manner the cutaneous area supplied by these nerves. The relations of this area to neighbouring areas are of considerable importance, and will be referred to later.

Herpes Zoster of the 3rd dorsal nerve with Hyperæsthesia.—Female, aged 13, has been under my care for over a twelvemonth for mitral disease, with symptoms of cardiac failure. On the 10th August, 1893, I was consulted because of an eruption that had broken out on the right chest. There was very little pain except on the inner side of the right upper arm. The eruption consisted of clusters of vesicles disposed as an interrupted band round the upper part of the chest and down the inside of the forearm. In front the clusters were continuous at their borders and represented a band about two inches broad. The most internal portion was situated over the sternum opposite the 4th costal cartilage. A few vesicles trespassed across the middle line for quarter an inch. From here the band extended outward and slightly upward, crossing over the 3rd rib to the front of the shoulder. A few scattered vesicles extended as high as the 2nd rib, and as low as the nipple—some being situated on the nipple. Behind the eruption extended as a somewhat broader band from near the spinal column, the centre being opposite the 2nd dorsal vertebra, outward and slightly downward over the scapular spine to the posterior axillary fold. On the right arm the eruption occupied the inner surface, two streaks occurring on the anterior and posterior axillary folds, and other two streaks occupying the anterior and posterior surfaces of the inner aspect about the lower third of the upper arm. Surrounding the area of eruption was a wide field of hyperæsthesia. In front it was as high as the second rib and crossed the middle line for at least one inch. Behind it ascended as high as the level of the 7th cervical vertebra and also crossed the middle line for a very short distance. It descended the side of the chest in an irregular manner as low as the 10th rib. It also descended the arm embracing the area of eruption. The interrupted line in fig. 2 shows the hyperæsthetic area.

The hyperæsthesia in this case extends somewhat diffusely, yet in a definite manner. The irritation from the diseased root that produces the eruption spreads to neighbouring nerve roots. But in doing so it evidently does not necessarily affect

the whole of one root before it passes to the next. Just as was pointed out in relation to the hyperæsthesia associated with visceral disease, contiguous fields are affected, without of necessity the whole area of supply of individual nerve roots being affected. The extension of the hyperæsthesia likewise takes place in a definite direction, not passing for instance over the clavicles into the field of supply of a distant nerve root, but descending on the arm.

A comparison of these two cases also shows a distinct overlapping of nerves. While it is difficult to prove this overlapping by the study of areas supplied by neighbouring roots, the opportunity is readily afforded when contiguous areas of skin are supplied by nerves whose roots are wide apart.

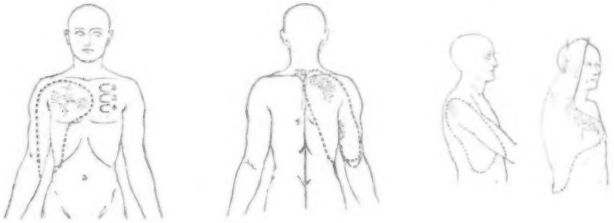


FIG. 2.

The interrupted line embraces the hyperæsthetic area.

Thus the skin of the upper part of the chest is supplied by the 4th cervical that descends over the clavicle as far as the 3rd rib in fig. 1, while the area supplied by the upper dorsal nerves ascends as high as the 2nd rib, as in fig. 2. This overlapping can also be seen at the back in these figures. This same spreading of the nerve supply of one field into that of another is also demonstrable in the crossing of the hyperæsthesia to the opposite side in fig. 2. The eruption has also crossed for a very short distance, and this was more unmistakably demonstrated in fig. 3.

I have been unable to find in my cases of herpes that accuracy of delimitation that Dr. Head describes. If the eruption in fig. 2 be studied, it will be found to occupy a fairly distinct field. The bands on the front and the back of the chest represent a fairly distinct continuity. But they

will be found to correspond to none of the single areas described by Dr. Head. As a matter of fact, the eruption trespassed into the areas that he reserves for the 1st, 2nd, 3rd, 4th, and even 5th dorsal nerves. Yet the continuity of the eruption suggests to me very strongly the idea, that there is but one nerve root affected here—in all probability the 3rd. In fact, I look upon it as being a more complete delimitation of the same field that his case 14, fig. 13, imperfectly represents. This is, I think, strikingly evident in the posterior band, where it cuts by a straight band the wavy contours that he depicts. In making my notes of this case I carefully copied the outlines of Dr. Head's fig. 13, in order that the comparison might be as true as possible. These copies made by me were repeatedly verified.

As a further illustration of the disagreement of my observations with those of Dr. Head in this exact delimitation of the field of supply by individual nerve roots, I give the notes of another case of herpes zoster under the care of my friend Dr. Harwood, to whom I am indebted for the opportunity of seeing the case, and for his kindness in verifying the particulars.

Dr. Head describes the lower border of the field of supply of the 12th dorsal nerve as sending two tongue-like projections down, one on the front of the thigh, and the other over the side of the haunch. These depressions dovetail into the field of distribution of the first lumbar. In a few cases I have certainly found the area of hyperæsthesia associated with disease of certain organs assume this shape. But that it represents the exact limits of the 12th nerve only is not supported by the symptoms in the following case.

Case of Herpes Zoster of first lumbar nerve.—Female, aged 50, suffers from attacks of excruciating pain round the right hip, where an eruption has broken out. The eruption extends from the vertebral column at the lower lumbar region round the right hip to the front and inner surface of the thigh in a band from four to five inches in breadth. Its superior border is extremely well defined, slanting in almost a straight line from the vertebral column at the level of the iliac crest, round the side of the ilium midway between the crest and great trochanter, to the front of the thigh

a couple of inches below Poupart's ligament, and almost parallel with it to the inner surface of the thigh. The lower border is not so clearly defined. It leaves the vertebral column at the level of the upper part of the sacrum, passes round over the ilium, and on the front of the thigh it is more irregular, sending scattered patches as low as the lower third of the thigh. The eruption distinctly crosses the middle line behind for at least the distance of one inch (fig. 3).

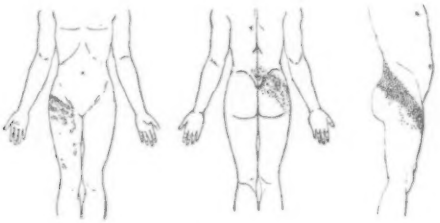


Fig. 3.

Here we have an eruption pursuing a fairly defined course, the upper limits of which slant in almost a straight line from the vertebral column to the inner surface of the thigh. Even if one assumes that part of the 12th dorsal root is involved in this case, it is surely too much to expect that the disease has picked out the portions of the roots affected, whose cutaneous distribution exactly fills up the gaps formed by the irregular outline of the 1st lumbar. Here again, there is undoubted crossing of the eruption to the left of the middle line, a further proof of the overlapping of the nerves.

From the study of cases of herpes zoster, and of the hyperæsthetic areas associated with visceral disease, I have come to the conclusion that there is distinct overlapping of the fields of cutaneous supply of individual nerve roots, of pathic, thermic, and trophic fibres, as well as of those of ordinary sensation. In this respect the results are in harmony with those arrived at by Sherrington from observation of isolated areas producing reflex contraction, with those obtained by Paterson from dissection of fibres from individual nerve roots, and with the observations of Langley on the pilomotor nerves.

VI.—Other Reflex Phenomena in Visceral Disease.

Besides the symptoms elicited from stimulation of the sensory nerve roots, a great number of other phenomena may arise from the stimulation affecting roots of nerves with different functions. If we consider that a stimulation arises in an organ and is transmitted by afferent nerves to a more central situation, and if this stimulation be of sufficient force or of the proper quality to affect certain nerve roots with which it comes in contact, then, according to the function of the nerve root, there will arise phenomena peculiar to the organ stimulated. Thus, if it be a sensory nerve root, there will be an exaltation of its function, giving rise to exaggeration of its peculiar property, or, it may be, causing a lowering of its functional activity, as when anæsthesia occurs. If the nerve root so stimulated be motor in function, then there may arise spasmodic contraction, or even loss of power in definite muscle groups, slowing of the beat or palpitation in the case of the heart. If the nerve root be secretory in function, there may arise an increased activity of the gland whose root is stimulated. In one remarkable case of angina pectoris I had under observation, the attack used to radiate characteristically from the chest to the left arm, and up into the neck. At the same time there used to be a striking and excessive discharge of saliva from the left side of the mouth. In another case of angina pectoris with aortic aneurism, where the pain was wont to radiate down the inside of the left arm, in this area, after a series of attacks, there was gradually developed a streak of ecchymosis, six inches in length. No cause could be assigned for its appearance, and I assumed it to be an affection of the trophic fibres. Hilton refers to certain trophic changes in the skin from disease of the teeth, and I have seen a case of ulceration of the abdominal skin after an attack of gall-stone colic, but as mustard had been used locally, I could not satisfy myself that it might not have something to do with the ulceration. Did we comprehend fully the manner in which this stimulation arising in a diseased organ spreads, we might be able more clearly to understand the meaning of many obscure cases of perverted functional activity of the various organs in the body.

VII.—Effects of Stimulation of the Skin upon the Viscera.

A certain amount of light is thrown upon the subject of the relationship between the cutaneous and the visceral innervation by the study of the effects of stimulation of the skin upon the viscera. The subject from this aspect is akin to that of the cutaneous reflexes, and doubtless lies at the foundation of the effects of treatment by counter-irritation, &c. There are certain well-known effects produced in the viscera by stimulation of the skin, and these are frequently taken advantage of in practice, *e.g.*, the counter-irritation in the pit of the stomach to allay vomiting, and the application of cold to the abdomen to cause firm uterine contraction. A very interesting light is occasionally shed upon this subject by the results of operations, as in the fact that after operations involving the skin of the perineum (for hæmorrhoids and for repair of torn perineum), not unfrequently retention of urine follows. Hilton had noticed that soothing applications to the perineum were beneficial in certain forms of retention of urine. Berry Hart [1] has recently noted a very striking instance of this relation between perineal skin and the muscles involved in the urinary reflex. Remarking the frequency with which retention of urine followed operations on the perineum, he determined to avoid the skin in his sutures. In a series of cases in which this plan was carried out the result was eminently satisfactory, as retention of urine no longer occurred.

The interest from the point of view with which I am dealing here is, that evidently the sacral nerves supplying the perineum are in association with the muscles of the bladder, and not of the mucous membrane alone as Head inclines to suggest.

VIII.—Practical value of recognising the Sensory Symptoms produced by Visceral Disease.

Treating the subject in this somewhat desultory fashion, it is difficult to convey properly the help that is afforded by enquiring carefully into these sensory symptoms in one's every

[1] Hart, D. B., "Perineal suture in relation to retention of urine after labour," *Edinburgh Hospital Reports*, vol. i., p. 587.

day work. I might be permitted to single out a few instances for the purpose of illustrating the value of those symptoms.

I have elsewhere called attention to the particular value of the seat of pain in intestinal obstruction. I do not see a sufficient number of cases to enable me to speak dogmatically on the subject, but in those I have observed, the results have been as striking as they were instructive. The pain in any given case of obstruction is due to the peristalsis of the bowel above the part obstructed. If, then, the pain is situated about the umbilicus, radiating more or less to one side, never descending lower, then one can infer that the point of obstruction is above the great intestine. If, however, the pain be felt as low as midway between the umbilicus and symphysis pubis, then some portion of the great gut is above the point of obstruction. In the latter cases the pain may arise in the umbilical (or even epigastric area) and gradually descend. It is necessary to see the patient when the painful peristalsis is upon him and to enquire carefully of him during the pain, as otherwise the information is too vague to be of service.

The observations that these hints are based upon, are from a dozen cases of hernia of the small intestines, and half-a-dozen cases of obstruction in some portion of the great intestine. The diagnosis was verified either by operation or *post-mortem* examination. These are certainly far too few observations to base an absolute statement upon, but they are of value as suggesting a help in the elucidation of a notoriously difficult point of diagnosis.

The sensory phenomena due to visceral disease may mislead very skilled observers. During my earlier observations on this subject I was called in consultation to see a man in failing health, in whom there was great pain round the left half of the body. There were remarkably few symptoms, besides the pain, and I found a hyperæsthetic area extending from the epigastrium round the left half of the body to the 6th and 7th dorsal vertebræ, which were extremely tender to pressure. I was puzzled with the case, and could only suggest that there was something wrong with the stomach. The patient receiving no benefit from my suggestions, consulted a skilled and deservedly eminent neurologist,

who, on the strength of the tender vertebræ and hyperæsthesia, diagnosed disease of the vertebræ, and recommended the encasing of the patient in a plaster of Paris jacket. This was done, with no relief. In course of time the patient died, and his medical attendant wrote me saying that he had had a *post-mortem* examination, and there was cancerous disease of the posterior wall of the stomach at the cardiac end, and the vertebral column appeared perfectly sound.

The following case illustrates how misleading the sensory phenomena may be in another way. I was called, in consultation, to see a young lady whom her medical attendant informed me was suffering from peritonitis. A year previous, while at a fashionable health resort, she was laid up for four months with peritonitis. The appearance of the patient was typical of that disease, the drawn face, the small quick pulse, and the flexed knees and high temperature. She shrank whenever we ventured to touch the abdomen. Accepting the case as one of peritonitis, in seeking a cause, I proceeded to palpate the region of the vermiform appendix. But the patient would not let me touch the skin, it was so painful. Testing with a pin-head, I found the skin in this region acutely hyperæsthetic, and I was able to map out an area roughly corresponding to the distribution of the 11th and 12th dorsal nerves on the right side. On careful exploration I found that the other regions of the abdomen were not really tender, her desire to protect herself from pain making her frightened of being touched anywhere. On enquiry into the state of the urine, I was informed it was all right, but on examination there was a small quantity of pus. On careful cross-questioning, a distinct history of severe spasms, prior to the last illness, was elicited. I therefore gave the provisional diagnosis of pyelitis. In a couple of days there was an abundant discharge of pus in the urine, the hyperæsthesia disappeared, the temperature fell, and the patient made a quick recovery from her anxious condition.

IX.—The Sensibility of the Subcutaneous Tissues.

A knowledge of the sensibility of the subcutaneous tissues of the external body wall would be of great value. Are the pains of tissues lying under the skin referred or local? It seems almost absurd to ask the question, yet there are a number of very curious facts elicited when the subject is enquired into—as the well-known pain at the knee from in-

flammation of the hip-joint, and the hyperæsthesia of the skin associated with joint disease. That the subcutaneous tissues can become acutely sensitive, has already been illustrated, and the occurrence of afferent nerve fibres in muscles has recently been put beyond doubt by the investigations of Sherrington. But when one comes to examine the matter it is very difficult to obtain satisfactory results. I have been in the habit for a long time of performing operations (after rendering the skin anæsthetic) without eliciting the slightest symptom of pain. Lately a number of hernias has been operated upon in the Victoria Hospital without an anæsthetic, and after the skin was cut no pain whatever was elicited. The last one, however, on which I operated, did not yield such conclusive results. In this case, when dissecting the tissues overlying the hernia, the patient occasionally called out. On testing the portion of tissue, the cutting of which had caused pain, I never could reproduce the sensation. After the hernia was reduced, I brought the deep parts together by a series of catgut sutures, until all the tissues, save the skin, were approximated. In such cases I do not suture the skin, but hold the edges together, and paint the line of incision with celloidin solution. This dries quickly and brings the skin edges firmly together. In this manner the whole of the tissues, from peritoneum to subcutaneous fat were systematically stitched, and there was not the slightest pain evoked.

In many ways the knowledge that subcutaneous tissues are practically insensitive to stitching is of great practical value. Thus in recent ruptures of the perineum, the torn surfaces can be brought at once accurately together, with practically no suffering on the part of the patient by avoiding the skin in suturing. When skin sutures are employed, the suffering is frequently so great that the perineum cannot be satisfactorily stitched without an anæsthetic.

These facts were matters of common knowledge in pre-chloroform days, and many surgeons of to-day are no doubt cognisant of them. In conversation with an aged surgeon, who had seen much practice before chloroform was introduced, he informed me that it was always understood that after the skin was cut no pain was suffered during an operation, unless occasionally when a nerve was cut.

THE SENSORY DISTRIBUTION OF SPINAL NERVES.

BY WILLIAM THORBURN, F.R.C.S.

Owing to its wide scope and the very special nature of the observations recorded in it, Dr. Head's paper[1] is one which it is impossible to discuss in detail, and subsequent research on his own lines can alone confirm, enlarge, or possibly in some respects correct his conclusions. My aim, therefore, in the following remarks will be rather to dwell upon those aspects of his work which come most closely into contact with some of the views which I have previously published; but in so doing I propose to take this opportunity of collating and arranging some of my own results, and of analysing the recent progress of our knowledge regarding the cutaneous sensory distribution of the spinal nerves. Before proceeding to do so we may, however, cast a glance at certain other important issues raised by Dr. Head's paper.

Briefly, Dr. Head may be taken to have demonstrated that: —

1. There are associated with various visceral diseases certain definite and constant areas of cutaneous tenderness, each area having a "maximal" region in which there is pain.

2. These maximal areas coincide with the areas which Mackenzie, Head himself, and to a less extent, other writers have shown to be the areas marked out by attacks of herpes zoster.

The conclusion drawn is that these areas represent the cutaneous distribution of the pain-appreciative or algetic fibres of each serial spinal segment, and as a corollary the distribution of the algetic fibres of each segment is thus deduced.

[1] Brain, Part I., 1893.

It is to the data of this important corollary that I propose mainly to refer, noting how far they either confirm or conflict with the deductions which had previously been drawn from other methods of clinical research, and the results obtained by Prof. Sherrington, from his experiments on monkeys.

The first consideration will be that of the value of the *method* employed by Dr. Head, and here we are at once met by the objection that areas of pain and of tenderness are extremely difficult to determine, in view of the essentially subjective nature of the record. For this reason they are *per se* less trustworthy than are observations of anæsthesia, and every clinician knows the difficulty of accurately defining an anæsthetic area. To the observations upon herpes, however, this objection is quite inapplicable, that disease depicting its area of distribution by the most definitely objective and easily recorded of signs. And as the two series of observations—those of pain-areas and those of herpes-areas—coincide accurately, we can only conclude that Dr. Head has overcome the difficulties of his first method, and that his areas are undoubtedly the areas of distribution of certain serial sections of the nervous system, although it remains to be proved whether these "sections" are spinal segments, nerve roots, or other serial arrangements.

Contrasting Head's areas with those obtained by clinical observation of anæsthesia, or with Sherrington's experimentally determined areas, we are at once struck by their sharp delimitation, and by the total absence of fading margins, and consequently of overlapping of adjacent regions in the series. Now there cannot be a doubt that the anæsthetic area resulting from disease or injury of a nerve root has a fading margin; that is, that the cutaneous distributions of the spinal roots overlap, often to a large extent. This want of sharp limitation is seen in cases of section of nerve roots, and in most cases of injury to the spinal cord (in which the nerve roots are usually crushed at the region of injury); and it is especially well indicated in Sherrington's experiments. Head himself explains the sharp limitation of his areas by supposing that they are the representatives, not of

the distribution of nerve roots, but of spinal segments, and he points out that visceral disease can only evoke cutaneous pain through the intermediary of such segments. This theory has thus an *a priori* basis of probability, and it is further in entire accord with what we know of the plexiform arrangements of the nervous system to suppose that, if there do exist such sharply defined spinal segments as are here assumed, each of them would have a connection with at any rate some adjoining nerve roots, and that thus the nerve root itself would represent not a pure segment but the mass of the fibres of its own segment *plus* some fibres from those above and below it. In this way we could at once explain the sharp definition of Head's segment-areas and the overlapping of the anæsthetic root-areas.

I believe also that there is some direct evidence that the spinal sensory segments are more sharply defined than are the root-areas. In most crushing lesions of the cord there is a coincident injury to one or more pairs of roots, which, arising above the level of the cord lesion, have not yet left the vertebral canal, and thus the upper level of the anæsthesia which we find in such a case represents not cord-anæsthesia but root-anæsthesia. In other cases the roots, being firmer and more resistant than the cord, escape injury, and our anæsthetic level is then truly that of cord-anæsthesia; and in penetrating wounds especially the cord may be divided without the roots being injured. Now I have for some time believed that when the roots were uninjured the anæsthetic line was more sharply defined than in other cases, *i.e.*, that "cord-anæsthesia," or "segmental anæsthesia" had a sharper boundary than "root-anæsthesia." This is a view to which I find it exceedingly difficult to apply the test of definite observations, because many considerations are required such as—Has hæmorrhage ploughed up the cord above the actual crush? Was the crush or other injury complete in the cord? And were the nerve roots really injured, or did they entirely escape? Owing to these difficulties we cannot draw certain conclusions from the notes of old cases which were not specially observed from this point of view, and I must therefore be contented with the expression of a personal

opinion that purely cord lesions give a more sharply outlined anæsthesia than do lesions which involve also the spinal roots.

In conclusion, it may be added that although I am strongly inclined to accept Dr. Head's view that his areas represent the cutaneous distributions of spinal segments, there remains to be disproved another explanation of his facts, viz.: that his areas are simply the *maxima* of the over-lapping root-areas as usually recognised and as illustrated by Sherrington. Taking such a view we might readily suppose that the maximal region of the sensory distribution of the root would be that affected by tenderness in an irritative condition, or by a "trophic lesion," such as herpes. In fact we actually find within Head's tender areas other smaller maxima, wherein tenderness rises to the level of pain, and whence herpes spreads to the remainder of the area.

Having said so much upon the general aspects of Dr. Head's paper we may now pass to the special consideration of the distribution of the various spinal roots, contrasting the results arrived at by different methods and different observers. The method adopted by myself has been that usually resorted to by clinicians, and its basis rests on the following facts:—First, we are able at once to determine by anatomical observation the distribution of certain roots, which have no plexiform connections, and which can thus be traced from their origin to their termination; such nerves are the first lumbar (dividing into the ilio-hypogastric and ilio-inguinal), the fourth sacral, and most of the dorsal roots; the lower level of the distribution of the cervical plexus is also well defined. Secondly, we have cases of disease and injury affecting known and limited regions of the cord, or isolated roots; by subtracting from the anæsthetic areas in such cases the areas whose nerve supply is anatomically known we arrive at the areas of distribution of the remaining nerves. All that is therefore necessary is a sufficient supply of cases and a clear localisation of the lesion, and, for obvious reasons, injuries are best adapted to our purpose, these having, as a rule, at least a well-defined *upwards* limitation. Thirdly, the results thus obtained can be checked

by observing the associated paralysis of muscles whose nerve root supply is known from other clinical observations or from such researches as those of Herringham, Paterson, Ferrier, and Yeo.

In the lumbar region, however, we are met by the difficulties that the whole of the nerves arise within a peculiarly small area, that the roots lie closely together in their long intra-spinal course, that the solidity of this region renders small and localised injuries infrequent, that the cases met with are few as compared with injuries of the upper part of the spinal cord, and, finally, that death rarely follows at a sufficiently early date to allow of satisfactory *post-mortem* observation of the primary lesion. For these reasons the lumbar roots are in some respects more difficult to trace than are those of the brachial plexus, but we may here derive much assistance from observation of lesions of the cauda equina, bearing in mind that, after a crush of the cauda, if certain nerves are paralysed (as regards either motion or sensation) whereas others are not, the injured nerves arise from lower roots than those which escape; and, when loss of function is partial only, such loss is more complete the lower the origin of the nerve root whose injury has given rise to it. The two last propositions, which I first published in "BRAIN" for 1888, are of general application; they have been of the greatest assistance to me in determining the distribution of lumbar and sacral roots, and have, I believe, been adopted by all subsequent writers. A more extended experience has now satisfied me that, in very rare cases, they may be departed from, and that they are therefore not absolutely reliable, but their general value has only been confirmed by subsequent cases, and Herter has since found an anatomical basis for the facts in the *post-mortem* observation that in a case of pressure on the entire cauda equina pathological changes were most marked in the lower roots.

Using the above methods and applying them to the study of a large number of cases of spinal injury, I was able to determine with some accuracy the sensory distribution of the roots of the brachial and lumbar plexuses, and the results thus obtained were published in various papers,

collated in my "Surgery of the Spinal Cord" (1889) and somewhat amplified in the "Jacksonian Essay" of 1890, which, although unpublished, is accessible in the library of the Royal College of Surgeons. Since writing the latter I have seen no reason seriously to modify the conclusions which were then presented.

As regards the brachial plexus, "the fifth cervical root supplies the region overlying the deltoid muscle and the outer aspect of the arm and forearm as far as the styloid process of the radius, or base of the thumb, and the eighth cervical and first dorsal supply the little finger and inner side of the hand, forearm and arm, the remaining roots providing for the central parts of the limb on both anterior and posterior aspects. The exact lateral extent of the bands of skin thus supplied by each root can hardly be determined, and is indeed probably not a fixed quantity; the boundaries are usually ill-defined, and there can be little doubt that vicarious conduction of sensory impressions readily arises" ("Surgery of the Spinal Cord"). These sentences are illustrated by the following diagrams:—Fig. 1 shows the upper level of the anæsthesia,[1] which was sharply defined, in a complete crush between the fourth and fifth cervical vertebræ, in which the intrinsic muscles of the upper limbs were totally paralysed; it thus illustrates the well-known lower border of the distribution of the cervical plexus. In fig. 2 we have the very common type in which the fifth cervical root is spared, the injury implicating the sixth and subjacent roots. In such a case the deltoid, biceps, brachialis anticus and supinator longus muscles would escape paralysis. Of this type many cases are recorded, but, as will appear from the diagram, the exact extent of anæsthesia on the ball of the thumb is not determinable. (Cases 3, 4 and 8 of my book are of this type.) In two other cases we had the condition represented in fig. 3, the whole of the intrinsic muscles of the arm being paralysed on the right side, whereas the deltoid, biceps brachialis anticus and supinator longus

[1] It should be stated that in the majority of the cases quoted sensation was tested only by the point of a pin, so that we are dealing rather with sensations of pain than with those of pure touch.

escaped on the left; thus then the fifth cervical root is spared on the left side only, and the difference in the anæsthesia of the two arms is well indicated. In this case the area of retained sensation barely reached to the styloid process of the radius.[1]

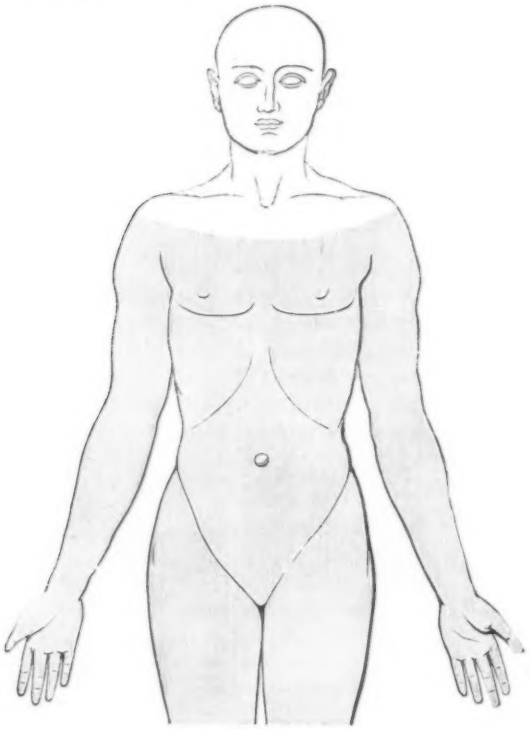


FIG. 1.

Turning next to the lower roots of the brachial plexus, we have cases such as those of which the anæsthesia is illustrated in fig. 4 (the dotted area was slightly hyperalgesic): in the patient from whom this drawing was taken

[1] For the sake of brevity the *post-mortem* examination of these cases, most of which have been elsewhere published, are here omitted. In all the cases quoted they accord with and explain the clinical condition.

the motor paralysis gradually extended up the cervical cord; on admission the intrinsic hand muscles and the flexors of the fingers and wrist were alone completely paralysed; on the second day the extensors of the wrist, and about the fourth day the pectoralis major, latissimus dorsi, teres major, and pronators succumbed. On admission "anæsthesia was

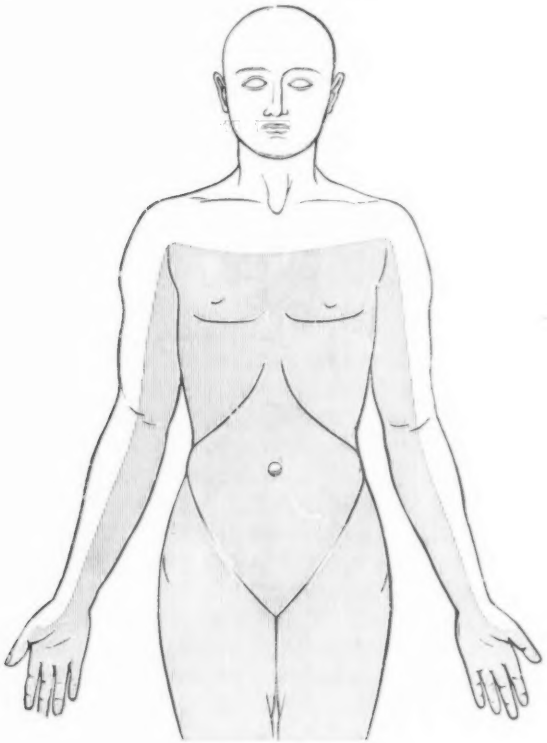


FIG. 2.

nowhere complete, but was almost so below the level of the third rib, and in the upper limbs internally to a plane running down the centre of the arms and forearms to the styloid processes of the ulnæ, both before and behind." The progress of the paralysis was followed by a gradual

increase in the intensity and extent of the anæsthesia, until ultimately the whole of both upper limbs were involved. (Case 10 in "Surgery of the Spinal Cord.") In this case the boundary of the anæsthetic area was never well defined, but in another patient (*loc. cit.*, Case 12), from whom is taken fig. 5, "in the upper limb anæsthesia was limited [with the

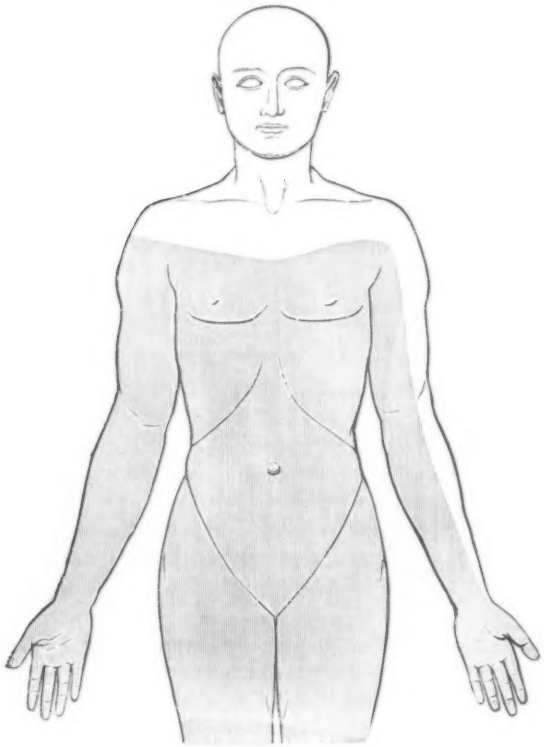


FIG. 3.

greatest accuracy to the inner side of the arm and forearm, being bounded in front by a line running vertically from the front of the deltoid opposite the second rib, down the centre of the biceps and forearm to the middle of the wrist; similarly behind the boundary was a vertical line down the

centre of the triceps and back of the forearm to the centre of the wrist. In the hand the limiting line sloped, both in front and behind, from the centre of the wrist to the cleft between the fourth and fifth digits, the little finger being completely anæsthetic, the others not at all so." Associated with this anæsthesia was total paralysis of the intrinsic

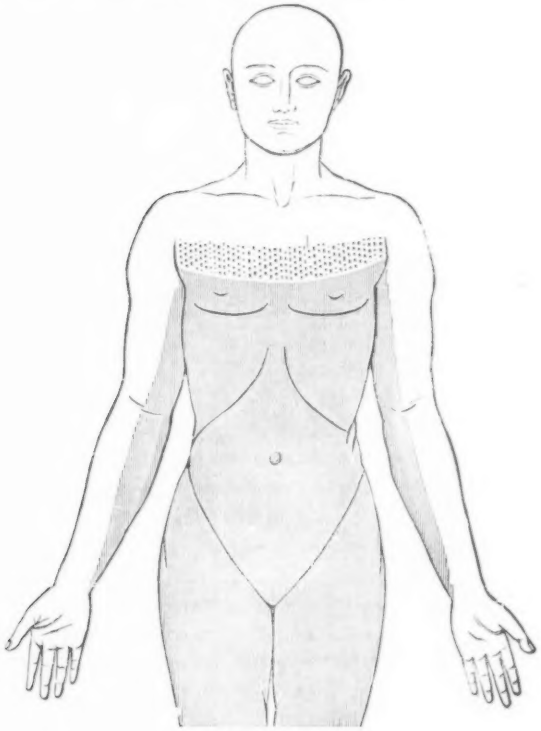


FIG. 4.

muscles of the hand, the interossei, and extensor communis digitorum, and great weakness of the flexors and extensors of the wrist, especially of the former. The lesion was a "fracture-dislocation" at the junction of the last cervical and first dorsal vertebræ, and had therefore probably implicated the eighth cervical, first dorsal and subjacent nerve roots.

From the above it would appear to me probable that the sensory distribution of the eighth cervical and first dorsal is represented by the anæsthetic area in fig. 5 and that of the first dorsal in fig. 4. Head (p. 44) has quoted the case depicted in fig. 5 as an example of anæsthesia resulting from injury to the first dorsal roots only, but a comparison with

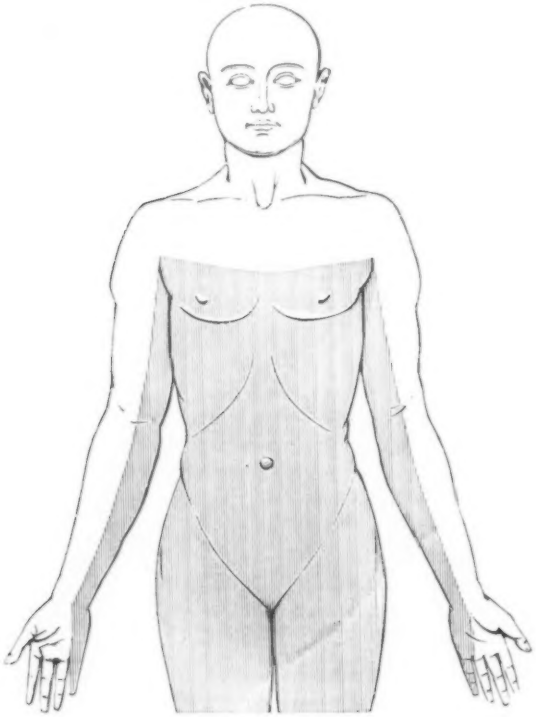


FIG. 5.

the previous cases leads me to the belief that the eighth cervical is here also involved. The above cases must suffice to illustrate the proposition with which the discussion of the brachial plexus was commenced: others have presented themselves to me since these were published, but they have not led me to modify the conclusions.

Turning now to the recent literature of the same subject, we find comparatively few observations, but such as have appeared confirm these views. Thus Starr (*Am. Journ. Med. Sci.*, May, 1888) gives the following in his table of the sensory distribution of the cervical nerves :—

5th. Back of shoulder and arm, outer side of arm and forearm to the wrist.

6th. Outer side and front of forearm, back of hand, radial distribution.

7th. Radial distribution in the hand ; median distribution in the palm, thumb, index, and one-half middle finger.

8th. Ulnar area of hand, back and palm, inner side of forearm.

1st dorsal. Inner side of forearm and arm to axilla. Some of these descriptions are difficult to follow without diagrams, but the coincidence of the two last with my diagrams is most striking. Bruns (*Deutsch. Med. Wochenschrift*, 1889) records two cases which bear out generally the above views. Mills (*Therapeutic Gazette*, 1889) gives Starr's table with modifications based on his own and other recent results, but in no way modifies the above quoted passages. Herter (*Journ. of Nerv. and Ment. Dis.*, 1891) records some interesting cases, and especially :—(1) Dislocation of sixth cervical vertebra ; softening of cord at level of eighth and to a less extent seventh cervical segment, " right arm, flexors and extensors of fingers and interossei completely paralysed, flexors of wrist very paretic ; left arm, flexors and extensors of fingers and interossei very weak, flexors of wrist slightly paretic ;" anæsthesia bounded by a line running down centre of arm and forearm, involving all the fingers and sparing the thumb and thenar region. (2) Crush of cord with serious injury to eighth segment, gradually returning to normal structure in sixth ; paralysis of intrinsic muscles of hands ; anæsthesia similar to last in arms and forearm but involving only the two inner fingers and the tips of the index and middle fingers. In both of these cases the anæsthesia is rather more extensive than in my cases with a similar amount of motor paralysis. Charcot records (*Archives*

de Neurologie, 1891) a case of injury to the first dorsal root on the right side, in which the anæsthesia was beautifully defined in the areas shaded in fig. 6, the immunity of the second dorsal distribution on the inner side of the first dorsal being also here shown; there was considerable atrophy of the hypothenar muscles and interossei, to a less extent of the thenar muscles, and some wasting of the forearm; paresis of the muscles of the hand and forearm, except the extensors; small pupil and small palpebral fissure. This case entirely confirms the views above expressed as to the first dorsal distribution.

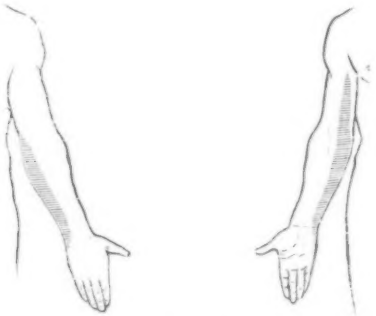


FIG. 6.

From Charcot, *Arch. de Neurologie*, 1891.

On comparing these areas with those obtained by Head's method, we find his first dorsal area to correspond very closely with that shown in figs. 4 and 6 of this paper, but Head's area is brought a little further on to the hand. This is the more remarkable as Head's areas tend generally to be smaller than the anæsthetic areas, and I cannot but suspect that his first dorsal region corresponds to at least a part of that supplied by the eighth cervical. And further, if we trace Head's areas downwards we find other evidences of a tendency to describe them as being a segment or part of a segment too high. Thus we find that he places the nipple in the area for the fifth dorsal root; whereas Sherrington states very definitely that, as a result of his researches, the

nipple (in the monkey) "lies in the middle of the fourth thoracic field," which corresponds to the fourth dorsal of man; and "Quain" has it that in man the anterior cutaneous branches "from the third and fourth nerves are distributed to the mammary gland."

If, however, we turn to the lower part of Head's diagram, there appears to be a tendency in the opposite direction, that is, the areas are depicted too low. Thus it will be seen immediately that his first lumbar region corresponds more closely to that which Starr and others besides myself assign to the second lumbar, and anatomical dissection shows that the first lumbar nerve is mainly distributed above the line of Poupart's ligament, where Head has the twelfth dorsal, and that only a portion of its area lies below the ligament on the front and inner side of the thigh. Again, anatomy does not indicate that the twelfth dorsal descends so far below the ligament as Head has it. And finally the umbilicus probably lies, not as Head places it, at the junction of the ninth and tenth dorsal fields, but certainly no higher than the lowest part of the tenth. Thus Rüdinger (*Anat. der mensch. Rückenmarks-Nerven*) shows the eleventh dorsal opposite to the navel; "Quain" (9th edition) places the umbilicus between the tenth and eleventh; and Schwalbe has it opposite the tenth.[1]

For these reasons I am inclined to believe that Head has carried his lower roots too low down, and his higher roots possibly too high up, and that he has therefore represented too many segments somewhere between the fourth and the ninth dorsal fields.

These discrepancies are rendered the more interesting by the fact that, when he comes to the lower lumbar and sacral roots, where his enumeration starts from a fresh point, his areas agree almost exactly with my own.

We may turn now to the *lumbo-sacral* nerves, but here space will not allow me to trace out the cases by which I have arrived at what I take to be the distribution of the

[1] For these anatomical references I am indebted to my friend Prof. Paterson, whose own authoritative opinion is that the umbilicus generally lies between the tenth and eleventh dorsal nerves.

individual roots. Most of these were lesions of the cauda equina and were highly complicated, but they are somewhat fully discussed in BRAIN for January, 1888, in the *Medical*

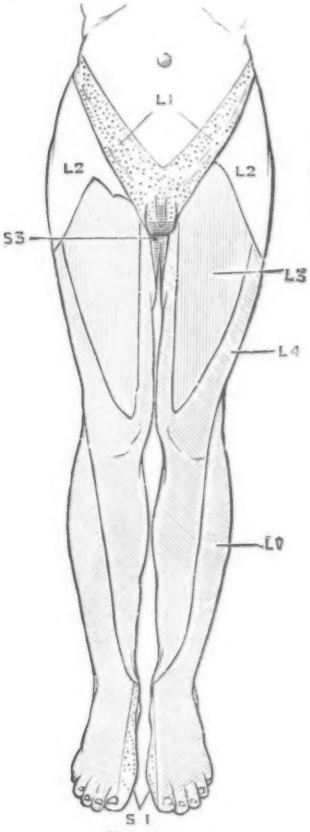


FIG. 7.

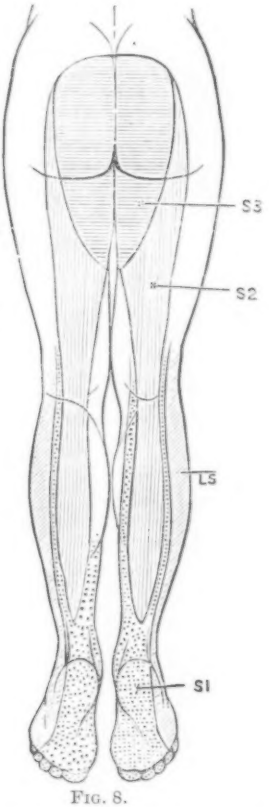


FIG. 8.

Fig. 7.—" L1, first lumbar; L2, second lumbar, &c.; S1, first sacral, &c."
Fig. 8.—" L4, fourth lumbar, &c.; S1, first sacral, &c."

Chronicle for 1889, and in my " Surgery of the Spinal Cord." On the basis of these cases I drew out a series of illustrations of the probable cutaneous distribution of each root, and these illustrations were collated by me in the Jacksonian

essay, although only verbally described in the published work referred to. They are now combined in a single diagram (figs. 7 and 8), where an attempt is made to illustrate the whole of the evidence derivable from cases of spinal injury. It must, however, be fully understood that the boundaries of the areas represented in these figures are not to be taken as absolutely defined; they are merely general delineations of the usual distribution of each root. Thus in the case of the sole of the foot it is not attempted to depict the true boundary between the first sacral and the fifth lumbar, there being no evidence upon this point among my own cases; and in certain cases where there were two conflicting results or series of results the two sides of the body are differently shown, as in the case of the second lumbar, which has at times a well-marked distribution on the front and inner side of the thigh, whereas in other cases it takes little part in the supply of this region. Lastly it must be added that the fourth sacral and subjacent roots are not indicated, being placed too low down in the perineum for representation in these sketches.

From the time of the publication referred to there date numerous records, all of which more or less closely confirm the results arrived at. Starr (*Am. Journ. Med. Sci.*, 1888) briefly records the results thus obtained without comment, and a year later Mills (*Therapeutic Gazette*) again endorsed them. The latter also publishes several cases of lesions of the cauda equina, which confirm the statements made above (*Medical News*, March, 1890, and June, 1889). Church and Eisendrath (*Am. Journ. Med. Sci.*, 1892) record a case which is peculiarly interesting, as there was a crushing lesion of the sacrum, cutting off some of the roots of the cauda equina whilst it spared others: such a case is of special value, as most of our evidence is derived only from general pressure lesions of the cauda, and we therefore reproduce the diagrams given by these authors (figs. 9 and 10). They state that the anæsthetic area embraced "exactly and only the cutaneous distribution of the four lower sacral nerves," but it will be found to involve at least a part of the territory assigned by both Head and myself to the first

sacral; at this we are not surprised on reading that upon operating for the relief of pressure "at the lower level of the first sacral vertebra the spinal canal was found, and the crushed-off termination of the dural sheath and cauda equina." As the first sacral nerves emerge below the first sacral vertebra we should expect them to be injured in a

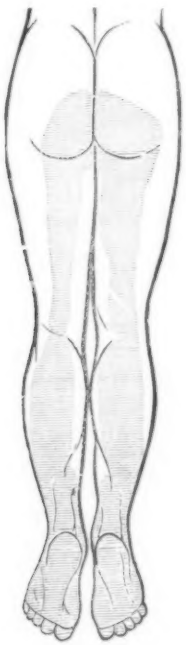


FIG. 9.

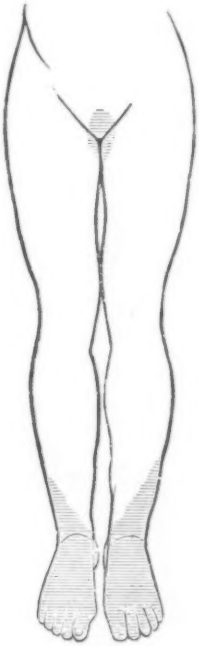


FIG. 10.

Figs. 9 & 10.—From Church and Eisendrath, *Am. Journ. Med. Sci.*, 1892.

lesion at its lower level, and we would suggest that Church and Eisendrath's case gives a picture of anæsthesia involving the first and subjacent sacral nerves.

Lastly Starr (*Am. Journ. Med. Sci.*, 1892) has recorded six original cases and collected others, in which localised anæsthesia of the lower limbs was produced by lesions of the cauda equina or conus terminalis. He gives a large

number of diagrams, which illustrate these cases in graphic form and, as a final analysis, he works out the distribution of several of the roots in the following figure, copied from his paper (figs. 11 and 12). This diagram differs very

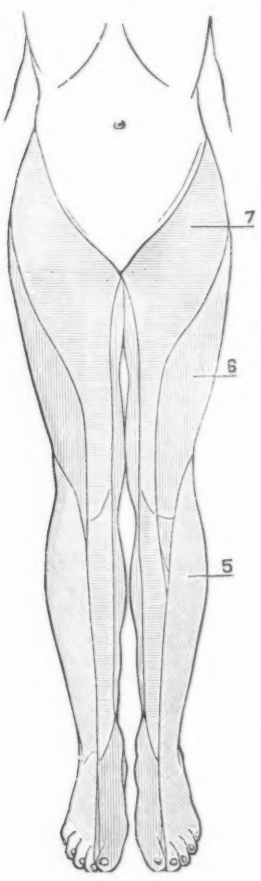


FIG. 11.

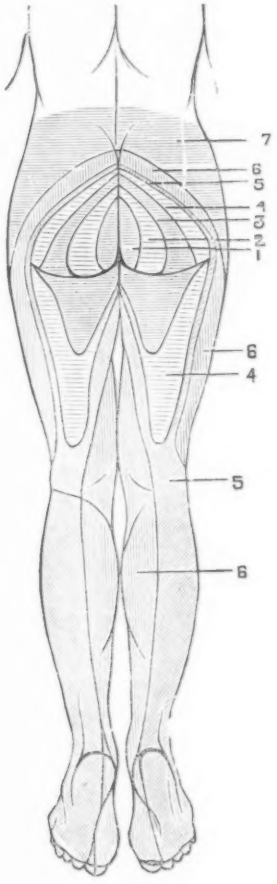


FIG. 12

1. Sacral V. 2. Sacral IV. 3. Sacral III. 4. Sacral I. 5. Lumbar V 6. Lumbar III. 7. Lumbar II.

Fig. 11.—From Starr, *Am. Journ. Med. Sci.*, 1892.

materially from both Head's and my own, but it is obviously not by any means perfectly correct, as the entire lower limb is furnished with root areas, whereas the fourth lumbar and second sacral nerves do not appear at all, and we can hardly doubt that these roots have sensory branches.

In conclusion, there is one more point to which reference must be made. It will be noticed that in my diagrams and descriptions, I have throughout indicated that the cutaneous area supplied by each root always extends upwards towards the trunk, tending to reaching to both mid-venter and mid-dorsum. The areas thus appear in the lower limb as a series of roughly-shaped V's and Y's, each being wedged as it were into that which lies next on its distal side. Thus fig. 7 shows the third lumbar splitting the fourth, and the latter again splitting the fifth; whereas in fig. 8 we have the third sacral splitting the second, and the latter dividing the first; this arrangement is less obvious in the case of the remaining roots, but the tendency to it will be observed in all. Similarly in the upper limb it was assumed that the sixth, seventh, and eighth cervical provided long narrow strips of sensation from the trunk to the hand, where they spread out to occupy a wider area.

The reasons for this view were partly theoretical, partly actual observation. The primary theory was that so fully explained by Ross, who regarded the limb as a bud thrust out laterally and carrying with it layers of cutaneous nerve supply derived from each of the segments to which it was related. Then again, the fact that the fifth cervical and first dorsal nerves supplied long narrow strips of skin appeared to render it probable that equally long and possibly narrower strips were provided by the intermediate roots. How far the influence of these views has biassed my observation of facts it is difficult to estimate, but I still incline to the opinion that, in the lower limb at least, we can identify long strips of cutaneous supply given by each root. Thus in the cases recorded by Kahler, Eulenberg, and Starr and Lloyd there is a very narrow band of anæsthesia down the back of the lower limb, a band which I take to represent mainly the distribution of the second sacral nerve. This band, which

unites the "saddle-shaped area," supplied by the third sacral, with the sole of the foot, supplied (according to Head and myself) by the first, has been several times observed by me, although it has always been in the form of a partial and not of a complete loss of sensation. Now I feel satisfied that this band is wider and its anæsthesia more marked if the anæsthesia extends to the first sacral or fifth lumbar nerves, and I have seen it become narrower and its intensity diminish when, during gradual recovery, these lower roots were regaining their function. This was well marked in Case 45 of my "Surgery of the Spinal Cord," although not clearly brought out in the diagrams—a remark which applies also to Case 32. The interpretation of this observation I have taken to be that the first sacral and fifth lumbar areas run down the whole length of the thigh on either side of the second sacral band.

Sherrington, on the other hand, in his experiments on the monkey, describes inter-digitating V-shaped areas similar to those in my diagrams, but denies the connection of the more distal roots with the mid-venter and mid-dorsum, and describes these segments as being "dislocated" by a lateral deflection into the limb of the mid-dorsal and mid-ventral lines.

It is perhaps too early to decide whether this view is entirely a correct one, but it must not be ignored that Sherrington bases his conclusions mainly on the fact that the hypothesis of an upward prolongation of every distal root area would imply a very great "overlap" in the limb. Although, however, the overlap would certainly cover many roots, it would involve only a very small cutaneous area, and it has yet to be proved that overlap does not depend at least as much on actual distance as on the number of root-areas covered.

ON THE EXACT SENSORY DEFECTS PRODUCED BY A LOCALISED LESION OF THE SPINAL CORD.

BY W. HALE WHITE, M.D.

THE following case seems to me worthy of record, for (1) it helps us to learn over what areas of skin sensation is perverted when there is a localised lesion of the spinal cord, and it is thus of importance in connection with Dr. Head's researches; (2) it illustrates what forms of sensation are the first to disappear when the cord is compressed, and it is therefore an aid to the elucidation of the paths of sensory impulses in the cord.

(1) The cord was compressed opposite the seventh dorsal vertebra, that is to say, at the point of origin of the eighth dorsal nerve. We ought, therefore, according to Dr. Head, to have found that the patient had loss of sensation below the upper margin of her eighth dorsal (middle epigastric) area. Reference to the figure will show that this was so on the right side. On the left there was loss of sensation below the upper limit of her ninth dorsal or supra-umbilical area. This difference on the two sides is probably to be explained by the fact of the cord not being compressed at exactly the same level on the two sides. There can, I think, be no doubt that this case strongly supports Dr. Head.

(2) We may from this patient perhaps learn something about the paths of the upward conduction of sensory impressions. The whole cord was compressed opposite the seventh cervical vertebra, but probably the lateral parts were much more compressed than the posterior, for on the sides of the cord there was some thickened inflammatory material which did not extend further back than the posterior roots.

In the first place, it will be noticed that over the area of skin corresponding to the whole of the cord below the point of compression, pain was interpreted as touch. This fact has often been observed before in similar cases, but nevertheless it is frequently overlooked, and cases are reported as suffering from cutaneous anæsthesia, when in reality they are only suffering from analgesia. Many authors assume that impressions of pain are different from those of touch, and that they ascend in different parts of the cord, founding this belief chiefly on cases such as the one here recorded. According to them, painful impressions are transmitted up in the lateral part of the cord, and simple sensations of touch in the posterior part. It is probable, however,[1] that "pain is no especial quality of sensation, but merely a special designation for the painful or unpleasant feeling that accompanies a very intense dermal sensation." On the first view we must assume that the pressure opposite the seventh dorsal vertebra so compressed the cord that it destroyed the functional activity of those fibres in it which transmit painful impressions; or on the second that those fibres in the cord which conduct sensations of touch were so compressed that their functional activity was lowered. This view is certainly supported by the fact that tactile sensibility was impaired—although it is true that the slightest touch was recognised—for the patient could not distinguish silk from velvet by the touch when they were applied to the feet, although a healthy person was able to do so, and with our patient it was necessary to remove the points of a pair of compasses before she recognised them as two, very much further apart than was necessary when the points of the same compasses were applied to the same parts of the skin of a healthy woman. But in favour of the first view is the fact that there is very strong reason for supposing that the lateral portions of the cord were more compressed than the posterior, and previously recorded cases of localised damage to the spinal cord appear to indicate that painful impressions are conducted up in the lateral columns, and those of touch in the posterior. I very

[1] Ziehen. "Physiological Psychology." Eng. trans., p. 133.

much doubt, however, whether in some of the previously recorded cases the statement that touch was unimpaired is as valuable as it seems, for observers have often been content with saying that touch was normal when the patient could feel the slightest touch. But our patient could do this, nevertheless it was shown by the silk and velvet, and compasses, that her touch certainly was not normal.

Over the whole of the analgesic area the patient could not distinguish between moderately hot and moderately cold objects applied to the skin, although she could between very hot and very cold. It has been observed in other cases of damage to the spinal cord, that when the capability of perceiving painful impressions, as such, is abolished, there is impairment or absolute abolition of temperature sense, and from them it has been inferred that sensations of pain and those of temperature are conducted in closely contiguous parts of the cord, and that as pain is probably transmitted upwards by the lateral columns, so probably are sensations of temperature.

It is especially interesting that both forms of muscle sense were unimpaired in this patient. She could tell an addition of 1 oz. in 12 oz. suspended from the ankle, and when I myself was tested I could do no better. Also she knew in what position her limbs were placed when she was blindfolded and I put them in any particular posture. These facts to some extent harmonise with the belief that the impressions of muscular sense and common sensibility ascend together in the posterior part of the cord, for this patient could feel the slightest touch, although in some particulars the sense of touch was impaired, and therefore on this view we must suppose that this part of the cord to some extent felt the effects of the compression.

The assignment of the conduction of pain and temperature to the lateral portions of the cord, and of muscle sense and touch to the posterior part, while no doubt it derives support from cases in which the cord was compressed from without, is difficult to understand when we study syringomyelia in which the pressure is exerted from the centre of the cord towards the periphery, for in them also

the power of appreciating sensations of pain and temperature is lost before the power of appreciating those of touch and those of muscle sense. But syringomyelia is often more marked at the posterior part of the cord than the lateral, so that we should have expected that sometimes at least the ability to distinguish tactile and muscular impressions should be lost before the ability to tell those of pain and temperature.

The last point to which I would direct attention is that there was no delay in perceiving sensations of touch or temperature, although both these were impaired, which seems to me very difficult of explanation, for we should have expected that if some fibres were so damaged that they could not conduct impressions accurately, neither would they be able to conduct at the normal rate.

On the whole, therefore, although at first sight this case supports the teaching that sensations of pain and temperature ascend in the lateral columns, and those of touch and muscle sense in the posterior, on looking more deeply we see some phenomena of it which are not explained on this hypothesis, viz.—the inability to tell silk from velvet, the wide separation of the compasses necessary before the points were distinguished as two, and absence of delay in the conduction of tactile and normal sensations. Facts like these should make us pause before assigning definite tracts to sensory impressions.

Sarah Pretlove, aged 54, admitted into Miriam Ward, Jan. 12, 1893. Clinical assistant, Mr. A. H. Leete, B.Sc. The family and previous history are unimportant.

Present illness.—This began three months ago with a pain which encircled the trunk on each side from a spot between the umbilicus and the ensiform cartilage. There was no vomiting, and the pain was not increased by food. About a month ago the patient noticed a few slight jerks in the leg, and that she staggered when she tried to walk. This increased till the present time when the patient drags the legs so that she cannot walk. She says that the legs always feel cold. Within the past three months a swelling has developed in the mid dorsal region of the spine.

Condition on admission.—The patient looks feeble and older than she really is.

Motor Symptoms.—There is no paralysis of the lower extremities, and the power of flexing the knee joints is good, and the thighs can be flexed on the abdomen, and the feet can be bent, but both lower extremities are in a typical spastic condition, and show the clasp-knife rigidity when extended at the knees. It is the spasticity which prevents the patient from walking.

Sensory Symptoms.—(1) Tactile Sensibility. There is no delay in perceiving touch, nor any impairment of the power of localisation anywhere. But there is some impairment of tactile sensibility, for the patient cannot by means of the soles of the feet distinguish silk from velvet, although it was found that a healthy person could. Further, the sense of touch (as tested by the minimum distance at which the points of contact of the points of a pair of compasses are perceived is two) is impaired thus :—

On soles of feet minimum distance ... ... = 1 in.
,, right leg just below knee ... ... = 1⅞ in.
,, left leg just below knee ... ... = 1⅝ in.
,, abdomen ... ... = over 3 in.

(2) Common Sensibility. There is no delay in sensation, but there is impairment, for if the patient be pricked with a pin she interprets the experiment as a mere touch. This analgesic area extends from below a line following the downward slope of the 9th left intercostal and the 8th right intercostal nerves down to the commencement of the lower extremities on both the back and front of the trunk (see fig.) The upper limit is absolutely definite on the front and sides. On the back it is slightly indistinct. It extends over the whole of the lower extremities except a few small areas where the prick of a pin gives rise to a sensation of pain ; there is one such area on the outer aspect of the middle third of the right thigh, and also one in a similar position on the left thigh.

No definite hyperalgesic area can be demonstrated, but it is probably not entirely absent. Attempts to locate it definitely lead to no constant result, but the results always lay between the 6th and 9th nerves. Pressure on the 6th right and 7th left intercostal nerves always caused pain. There is a very definite and constant girdle pain encircling the body. When asked to localise it the patient always places her hand over the epigastric and hypochondriac regions. The pain she describes is always present, and is of a dull heavy character.

(3) Temperature. There is no delay in perception, but over the whole of the analgesic area there is impairment of power to distinguish between heat and cold, but the power is nowhere

absolutely lost, nor is it ever perverted. On the chest and arms above the analgesic area there is no impairment.

(4) Muscle Sense. The patient when blindfolded can always tell the position her lower limbs are in when at rest, and also the position in which an observer may place them. When a broad bandage is placed round the ankle and a weight suspended from it, the least increase the patient could detect on either side was 1 oz. added to 12 oz.

Reflexes. The knee jerks are increased, the plantar reflexes are lively, the epigastric reflex is absent. Ankle clonus is present.

Micturition and defæcation are normal. There are no trophic changes.

Spinal Column. There is a considerable backward projection of the 7th to 10th dorsal spines, which form a prominent convex swelling.

The respiratory, circulatory, alimentary, urinary systems are normal.

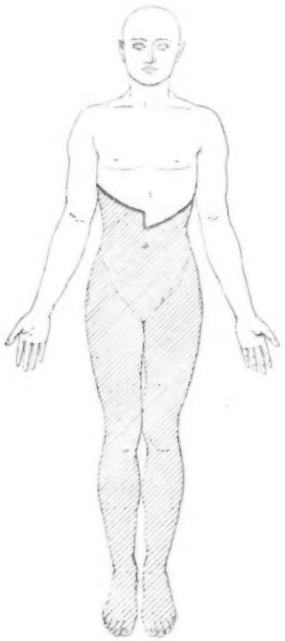
January 20.—Mr. Lane cut down upon the swelling in the back. The laminæ of part of the 6th, of the 7th, and part of the 8th vertebræ were removed. The cord did not pulsate. A spoon was introduced between the bone and dura mater, and pus welled up on either side of the cord, and on thrusting a probe between the 6th and 7th dorsal vertebræ much more pus welled up and some pieces of carious bone came away. A channel was then scraped between the two vertebræ, and an abscess cavity on the front of the spine was detected. This was thoroughly syringed out, and much pus flowed away. The cord commenced pulsating when the pus was let out. Drainage tubes were put in, the edges of the wound were brought together, and it was dressed with cyanide gauze.

January 23.—The patient, who has never thoroughly rallied from the operation, gradually sank and died at 10 p.m.

January 24. *Autopsy.*—In the chest on each side of the vertebræ was an abscess cavity ; on the right side it was in contact with the 6th, 7th and 8th vertebræ, on the left with the 7th and 8th. These abscess cavities extended laterally from the vertebræ for about two inches under the pleura. The 5th to the 8th vertebræ were removed for examination. The body of the 7th vertebra was almost completely destroyed, the only living part of it being a small fragment attached to the 8th vertebra. The posterior third of the intervertebral disc between the 7th and 8th vertebræ was destroyed. The disc between the 7th and 8th was intact. There was no pus within the dura mater. The posterior

part of the cord appeared quite normal. On the lateral aspects of the cord there was some thickened inflammatory material which extended to the posterior margins of the posterior roots, but, as just mentioned, left the posterior part of the cord free. Anteriorly it had extended over the cord, but to a less extent than laterally. Opposite the 7th vertebra the cord was soft and compressed.

There was healed phthisis at the apex of each lung, and there were scars of old gastic ulcers in the stomach.



DESCRIPTION OF FIGURE.

Over the shaded area and over a corresponding area at the back there is:—

(1) Analgesia. Pain being interpreted as touch.
(2) Impairment of tactile sensibility: (*a*) velvet not distinguished from silk; (*b*) points of compasses far apart called one.
(3) Impairment of power to tell hot and cold apart.
(4) No delay in perception of pain, touch or temperature.
(5) No inability to tell passive position of limbs.
(6) No inability to tell added weights.

ANGIO-NEUROTIC ŒDEMA.

(With a record of five cases.)

BY ERNEST WILLS, M.D., M.R.C.P.LOND.

AND

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THE occurrence of various transient vascular phenomena of the nature of flushings, erythema, urticaria, acne rosacea, &c., in dyspeptic and hysterical patients, as well as in girls at the menstrual periods and women at the climacteric, has long been recognized. The appearance of "Œdema in a limb, the subject of hysterical paralysis, and its disappearance with the recovery of power have, since the publication of Charcot's lectures, been admitted to be of neurotic origin." Bauke last year drew attention to the occurrence of circumscribed œdema of sudden onset and rapid disappearance in individuals of neuropathic tendencies without other signs of disease, and gave details of two cases. We record here five cases, in all of which the patients were free from any organic disease which would have had any influence at all on the production of this condition. In no case was the temperature raised, the œdema was painless in character and did not disturb the general health. An œdema of probably purely nervous origin is comparatively rare, and we have therefore thought these cases worth recording, and have offered some remarks on their general etiology and pathology, and have indicated the position they seem to us to occupy among the neuroses.

Case 1.—Peter M., aged 22 years, was under the care of one of us in the wards of Highgate Infirmary for compression paraplegia.

The patient was very neurotic, and from time to time had attacks of hysteria. He was probably a masturbator.

The following notes were taken during an attack of the transitory œdema we are now describing:—

April 19, 1891. A circumscribed erythema has appeared on the face around the right eye. There is a slight pitting on pressure, and a rise of local temperature perceptible to the hand. Temperature in rectum, 98.6.

Later in the day a redness with œdema was noticed over the left shin.

April 20. Erythema fading. Œdema gone.

April 21. There are swellings similar to those described above to be seen on both sides of the neck and on the back of the first phalanx of the right thumb.

April 22. The swelling has disappeared from the thumb, but the back of the hand and wrist is red and swollen. Towards evening the under lip became swollen, and at midnight there was a brawny red swelling on the back of the neck. None of the swellings are at all painful to the touch, and the patient was once or twice unaware of the swellings until they were pointed out to him.

The temperature taken every six hours was normal throughout the attack.

April 23. The patient is very hysterical, alternately laughing, singing, crying, and praying. He refused food for thirty-six hours. All trace of œdema and erythema has disappeared.

Early in May the patient's tongue became swollen. This swelling lasted eight or nine hours. He was then quite well for some months.

Case 2.—Ellen N., aged 14 years, a pale, nervous, and excitable child, was under observation in November, 1891, complaining of pains in the back and a feeling of general malaise. She drew attention to a red and œdematous swelling on her right elbow which had appeared that morning. Pressure on the swelling caused no pain. Patient stated that she had had a similar swelling over her forehead the previous evening, of which no traces remained. The patient was seen four days later and said that the swelling on her elbow had disappeared on the evening of the day on which she had first noticed it, and that it had been followed two days later by a swelling of the same nature on the back of the right hand, which also had disappeared in the course of a few hours. When seen again there was a distinct circumscribed œdema, with redness over the left ankle, hot to the touch,

but not tender. (Temperature in mouth, 98.8.) One week later the catamenia appeared. After this no more local swellings were noticed, and the patient returned to her former good health. She was seen again six months later, and up to that time had kept quite well.

The following cases came under observation at the County Asylum, Rainhill, and we are indebted to Dr. Wiglesworth for his kind permission to publish them :—

Case 3.—James McG., aged 22, single, labourer, Irish, was admitted April 16th, 1892, suffering from adolescent insanity. His father, an excitable neurotic looking man, said that patient was the fifth of a family of eight and had always been "dogged and stubborn." Two of his sisters died of "decline."

On admission all his organs and urine were normal. He had widely dilated but reacting pupils. Ophthalmoscopic examination revealed no abnormalities. His palate narrow, somewhat pointed and high arched of "neurotic" type. Owing to his mental condition it was impossible to investigate satisfactorily his sensation and special senses. No anæsthesia was made out and no hysterogenic points ; he certainly stood pricking all over remarkably well, and seemed to take a positive pleasure in protruding his tongue and having it deeply pricked. He was quite aware of all that was done to him, and though pretending to be uninterested he furtively watched all movements made during his examination, very much after the fashion of a hysterical girl.

When left alone he would sit motionless with head bent and eyes fixed on the ground ; he would push away anything placed to intercept his gaze. He was generally neglectful of himself, dirty in his habits, and resistive.

He remained in this condition, with little change, for six months, when he was sent to the infirmary because he stood about in the cold with blue extremities and deficient circulation. Soon after this he became actively mischievous and dirty, and would sit about in contorted positions, such as "with his feet and legs held rigidly in front of him and his arms held out from his side horizontally," or "with the foot held by his two hands and thrust into his mouth," for half-an-hour or more.

October 24. His face was noticed to be swollen, somewhat red, but not inflamed, and there was œdema under the eyes and slight pitting on pressure over bony parts. There was no rise of temperature, which was taken every four hours, no albuminuria, no periostitis or dental trouble.

October 26. Swelling and œdema are little altered and have not extended beyond the face. There is no œdema or swelling in any other part of the body. Urine normal. Patient was kept on light diet for a few days, and the œdema gradually passed off, leaving slight pigmentation, but no other trace. The general health was unaffected. There was no erythema or urticaria.

December 18. Patient became very troublesome and mischievous, continually twisting his head round, biting his shoulders and arms; he would get out of bed at night and gnaw the linoleum on the floor. Swelling of his face with some reddening and œdema was again noticed, for which no cause could be found. His face felt hot, but there was no general rise of temperature and no œdema or eruption on the body. The œdema passed away again in a few days.

February 28, 1893. There have been no further attacks. He has changed little mentally, although a little more rational, and moving about when ordered, he is still fond of assuming *bizarre* positions, and conducts himself generally in a hysterical manner. We would draw special attention to this hysterical condition in both Cases 3 and 4, as having an important bearing on the causation of the vaso-motor trouble.

Case 4. George H., aged 22, single, tailor, was admitted May 22, 1892. A maternal aunt died in Rainhill asylum, of general paralysis. His brother said:—Patient was never a very bright lad but got on fairly well at school, which he left at 16, and worked at tailoring. Twelve months before admission he had an attack of influenza, which began with faints and severe pains in the head. He was laid up for a fortnight. Soon after this he became excited and talkative, and was continually preaching. After being three weeks in the workhouse infirmary for his mental condition he was still dull and stupid, and was sent to the country for a change, but did not improve, and has remained the same until now, except for occasional outbreaks of violence and excitement.

On admission there was no organic disease. Ophthalmic examination revealed no change in disc or fundus. His pupils were dilated, but reacted to light. His tendon reflexes were very exaggerated. He talks about religion and prays, but is incoherent, irrational and flighty, with a furtive smile and very self-conscious expression.

June 10. Patient is without doubt a masturbator, his mental condition is very variable, he is sometimes stuporose, at others restless and mischievous.

October 29. He was sent to the suicidal ward that he might be under continual supervision. He had threatened to throw himself out of the window; he was continually pulling at his finger nails and had torn two completely off. He had more than once tied his scarf tightly round his neck. He has picked some sore places on his head which are raw and granulating from continual irritation.

February 3. He has œdema and flushed face. There is swelling under the eyes and slight pitting on pressure over the frontal bone. On examination, his chest, abdomen, and the upper parts of the arms and legs are seen to be covered with a diffuse bright red punctiform rash resembling that of scarlet fever. The redness fades on pressure, but there is no œdema of any part of the body. Slight red lines arise after stroking the skin with the finger nail and remain visible for some minutes. No rise of temperature, no albuminuria, no increase of redness or swelling around the sores on the vertex.

February 5. The urticarial eruption has almost disappeared from the body and limbs, but an *artificial urticaria* can still be produced. The œdema of the face is less marked.

February 10. Œdema and urticaria completely disappeared.

February 20. Slight flushing and erythema of face. No œdema. A kind of *tache cérébrale* can still be obtained on the body.

His knee jerks are still very exaggerated and a patellar clonus can be most easily obtained, which is accompanied by a general tremor of the body almost approaching a rigor. With a little encouragement and suggestion no doubt a hysteroid fit might be induced.

February 28. No recurrence of œdema. He is in good general health and condition. We would add that influenza, appearing as an antecedent in this case, is becoming very frequent in the history of insane patients as an exciting cause.

Case 5.—Mary G., age 47, widow, charwoman. She has had three children, no miscarriages. Her husband died twenty years ago. She has been twice in Chester Asylum, for periods of three and four months. She was admitted December 2, 1892, suffering from dementia, secondary to climacteric insanity. Organs and urine normal; no skin trouble. Says she has not menstruated for six years. No signs of syphilis.

February 18. She has an erythematous swelling of the forehead and temples, extending slightly to the face, pitting on pressure, and feeling hot to the touch; there is also an erythema

of the chest and abdomen, rather wider in distribution on the right side than the left. There is no hyperæsthesia or pain. No general rise of temperature. The urine was not examined. The erythema and œdema disappeared in a day or two, and the patient returned to her work in the laundry.

February 25. A slight patchy erythema without infiltration or œdema was noticed on the temples, and a patchy erythema of the neck and chest. On the abdomen in the hypogastric and neighbouring lumbar regions was found an œdematous and reddened condition of the skin, somewhat irregularly distributed, the margin of the affected portion being fairly sharply defined. The whole area affected was an irregular surface of about 12 by 10 inches. Temperature taken in rectum, 99.2. Patient complained of slight headache and a "feeling of water rushing up and down in the head."

February 26. Infiltration of skin of abdomen disappeared, leaving the skin slightly loose and wrinkled and a little pigmented. No erythema remaining in the abdomen. Slight traces of erythema remaining in the face. Urine, sp. gr. 1015, acid, a trace of albumen, no sugar, no casts, no lithic deposit. Temperature normal. Says she feels better in her head, but "the water still rushes up and down."

March 1. No remains of erythema or œdema. She feels quite well.

No friends have visited this patient, so that no history has been obtained, and the presence of heredity in this case is uncertain. The patient never heard of any of her friends ever having been in an asylum.

Etiology.—Numerous observers, for the most part continental, who have noticed this condition of fugitive œdema have implied their opinion of its origin in such terms as "nervous," "angio-neurotic," "neuropathic"; and we ourselves are convinced of the evidence of a strong neuropathic taint in all our cases. We therefore think that among the chief predisposing causes should be placed hysteria, hystero-epilepsy, neurasthenia, and the allied emotional states. Quincke, Strübing, Valentin and Osler think that heredity is an important etiological factor, and we have been able to obtain histories of a nervous disease although not of a similar affection in two of our cases.

First among the exciting causes, we would place psychical

disturbances, and in this opinion we are confirmed by numerous authorities such as Bauke, Charcot, Küssner Bitot, E. Wagner, Napier, Richt, and Lesser.

Max Joseph, who records three cases of this affection, thinks that alcoholism plays a prominent part as an exciting cause. In his cases, all of whom were dipsomaniacs, abstinence from alcohol brought disappearance of the swellings, but when excesses were again committed the œdema reappeared. Other exciting causes we would instance are—(i.) The onset of puberty—Case 2 and Börner. (ii.) The climacteric—Case 5 and Börner. (iii.) Gastric irritation—Cases 3 and 4 and Strübing. (iv.) Exhaustive nervous drain as masturbation, &c., during the unstable time of adolescence—Cases 3 and 4.

Cutaneous distribution.—As will be seen by references to the clinical cases recorded, the skin-distribution of angio-neurotic œdema, like urticaria and unlike herpes zoster and some forms of psoriasis, is irregular.

Pathology.—That the vaso-motor system is greatly affected by emotional states is well known; the phenomena of blushing, flushing, and sudden pallor are so familiar to us all as to be looked upon as indications of certain normal mental conditions. But in any morbid change in the mental state such as is met with in hysteria and hystero-epilepsy, the influence which the vaso-motor system of nerves has normally over the calibre of the arteries is markedly increased. Examples, of course, are daily met with in the pale skin of an hysterical girl which does not bleed on pricking, and in the flushings and sweatings that are seen in women at the menopause. It is only to be expected then that as we ascend the scale in abnormal emotional states so we find stronger evidence of loss of control over, or greater irritability of, the vaso-motor system. It being admitted that neurotic hyperæmias exist, the question arises as to whether the dilatation can be carried a stage further, still under purely nervous influences, and a neurotic œdema be produced. We venture to think that this question may be answered in the affirmative if both clinical and experimental facts be taken into account.

Taking first into consideration what we believe to be clinical evidence in support of the view we advance.

Gowers has recorded a case of trigeminal neuralgia in which all the veins of the affected side of the face became distended during the paroxysm, and another case in which cranial neuralgia was accompanied by an œdematous swelling of the whole scalp. In cases of neuralgia Anstie has noticed a general arterial dilatation during the paroxysm, which he has demonstrated by sphygmographic tracings. Numerous cases of vaso-motor disturbances following diseases of the spinal cord are on record. Gowers states that they are chiefly seen in cases of disease of the intermediate grey matter and Leyden has noticed considerable œdemas develop very rapidly in cases of acute myelitis. Crocker mentions the case of a lady "in whom the advent of strangers produced an urticaria, and this sensitiveness increased until a knock or a ring at the front door would determine an outbreak." This case is of extreme interest as an example of the great irritability of the vaso-motor system in the emotional states, and we shall discuss later the near relationship that some forms of urticaria bear to angio-neurotic œdema.

From the experimental point of view also, evidence is not wanting of the existence of œdema caused by nervous changes. Ostroumoff produced œdema of the tongue of a dog by irritating the peripheral end of the divided lingual nerve by induction currents of gradually increasing strengths, and Gergeus observed that frogs whose spinal cords had been destroyed became œdematous under certain conditions.

But a still more important experiment is that of Ranvier who ligatured the vena-cava on one side and did not obtain venous stagnation in the limb until he had also cut the sciatic nerve on that side. It is fair to say that Sonitschewsky differs from Ranvier, and states that in cats and rabbits very slight œdema indeed results from this operation. But Ranvier's work has recently been repeated by Horsley and Boyce, and they obtained the same result as Ranvier in nine out of twelve dogs experimented upon.

Finally, Tigerstedt and Santesson have given practically conclusive evidence that the transudation of living fluids through living membranes is not a mere physical phenomenon solely due to changes in pressure.

We think, therefore, that we are justified in assuming that the case for an angio-neurotic œdema is fairly made out —that is to say, that in a certain class of cases an œdema occurs which is due to a direct influence of the nerves on the vessel-walls causing an increased permeability. And we also think that a distinction may be drawn between the œdema of neurotic origin, which is almost certainly due to an increase of nerve excitation, and the œdema such as was observed by Gergeus in paralysed frogs, which is very possibly due to a cutting off of the vaso-motor nerves from their centres, or in other words, to a loss of vascular innervation. The sequence of events in the production of this neurotic œdema is probably as follows:—A local paralysis of the vaso-constrictors or a reflex stimulation of the vaso-dilators, causes a dilatation of the vessels of the subcutaneous tissues, which is followed by retardation and stasis of the blood. An exudation, usually serous, sometimes sanguineous, then occurs, producing an œdema, but the œdema is not sufficiently acute or sufficiently localised to cause any lifting up of the eperdermis as a whole, and the resulting condition may be fitly termed an *abortive urticaria*.

Quincke, Dinkelacker, Rapin, Minich, and Osler have all noticed cases in which this condition of fugitive œdema was present, and in which there were subsequent attacks of genuine urticaria.

Strümpell also remarks on the near relationship that angio-neurotic œdema appears to bear to urticaria, and his view of the pathology of the former affection is that it is a transition stage between a vaso-motor and a vaso-trophic disturbance. In all the cases now recorded by us it will be observed that a local stimulation produced a marked vaso-motor change—sometimes merely a *tache cérébrale*—at others a factitious urticaria, and in one case a genuine urticarial eruption was met with in another part of the body.

It has been suggested that in urticaria the formation of the wheal is the result of the contraction of the muscles of the skin, and it is just possible that in angio-neurotic œdema

no wheal is formed owing to the want of tone of these muscles. Considering the neurasthenic state of the majority of the patients in which angio-neurotic œdema occurs, such a loss of tone could easily be conceived.

Another point in common between angio-neurotic œdema and urticaria is transient albuminuria. Lente has noticed this in the latter affection, and we have observed it in Case 5.

Diagnosis.—This is, as a rule, a very easy matter, especially if its intimate connection with urticaria be borne in mind. Some clinicians, however, seem to have confounded this affection with the non-febrile form of erythema nodosum—for instance, Bitot and Simon have noted tenderness of the swelling, which is never present in neurotic œdema, and Küssner has described it as occurring with a rheumatic attack. On the other hand, Lewin, Auspitz and Schwimmer, have stated their opinion that the non-febrile form of erythema nodosum is due to a vaso-motor disturbance.

In conclusion, we have endeavoured to shew that the symptom is one of the vaso-motor neuroses, and the importance of recognising it lies in the fact that it may be an additional guide in the diagnosis of neurotic cases, since it is an index of an unstable nervous system. Measures for its treatment should be directed towards the central nervous system after all sources of possible peripheral irritation have received attention.

REFERENCES.

Bauke, H. "Zur Ætiologie des 'Acutes, Angio-Neurotischen' oder umschriebenen Hautodem," *Berliner Klinische Wochenschrift*, No. 6, 1892.

Bitot, Emile. "Les troubles trophiques dans l'Hystérie," *Progrès Medical*, xiii., August, 1891.

Borner. *Volkmann's Sammlung Klinischer Vorträge*, No. 312, June 11, 1888.

Charcot. "Blue Œdema in Hysteria," *Journal de Paris*, iii., August, 1891.

Clouston. "Neuroses of Development," 1891.

Cohnheim. "Lectures on General Pathology," Leipzig, 1882.

Crocker. "Diseases of the Skin," London, 1888.

Dinkelacker. "Inaugural Dissertation," Kiel, 1882.

Gergeus. Quoted by Cohnheim.

GOWERS. "Diseases of the Nervous System," 1886.
HORSLEY AND BOYCE. "A Preliminary Report on Œdema," *British Medical Journal*, January 21, 1893.
JOSEPH, MAX. "Ueber acutes, umschriebenen Hautödem," *Berliner Klinische Wochenschrift*, 1890, Nos. 4 and 5.
KÜSSNER. "Ueber hydropische Auschwellungen unklaren Ursprungs." *Berliner Klinische Wochenschrift*, 1889, No. 16.
LESSER. "Lehrbuch der Haut-und Geschlechtskrankheiten," Bd. i., s. 102.
LEWIN. *Berliner Klinische Wochenschrift*, No. 23, 1876, and *Neue Charité Annalen*, iii., p. 62.
LEYDEN. "Klinik der Rückenmarkskrankheiten," ii., p. 173.
MINICH. Riv. venet. d. x. med., 1887, i.
NAPIER. *Glasgow Medical Journal*, April, 1889.
OSLER. *American Journal of Medical Science*, April, 1888.
OSTROUMOFF. *Pflägers Archiv.*, xii., p. 219.
QUINCKE. *Monatschrift für pract. Dermatol.*, 1882, No. 5.
RANVIER. *Compt. rend.*, lxix., No. 25.
RAPIN. *Revue méd. de la Suisse rom.*, 1886, Nos. 11 and 12.
SCHWIMMER. "Neuropathischen Dermatosen," p. 101.
SIMON. "Acute Angio-neurotic Œdema." Reports of John Hopkins University.
SONITSCHEWSKY. *Virchow's Archiv*, Bd. lvii., p. 85.
STRÜBING. *Zeitschrift für Klinische Medicin*, 1885, Heft 5.
STRÜMPELL. Lehrbuch der Speciell. Patholog. und Therap., ii., Bd. i., Th. S. 130.
TIGERSTEDT AND SANTESSON. *Mittheilung vom. Physiolog. Laborat. des Carol. Med. Chir. Inst. in Stockholm*, 1886, and *Bihang vid. k. Svenska Sed. Akad. Handlingar*, Bd. xi., No. 2, June 6, 1885.
VALENTIN. *Berliner Klinische Wochenschrift*, 1885, No. 10.
WAGNER, E. *Archiv für. Heilkunde*, Band xi.

THE RELATIVE IMPORTANCE OF THE MINUTE HISTOLOGICAL FEATURES OF THE BRAIN CORTEX IN GENERAL PARALYSIS.

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THE perennial interest of general paralysis must be my excuse for a short essay on the minute pathology of a morbid condition which has engrossed the attention of the best observers in psychological medicine both here and in other lands. It is not my intention to go over and describe once more the morbid changes patent to the naked eye of any observer; nor even to give a detailed and complete account of the appearances which have been recognised under the microscope, and described with a fulness and accuracy not to be improved upon. The exact significance of the changes has been, and still is a matter of dispute. The relative importance of the pathological features of the different structures of the brain cortex still furnishes a field for investigation, and the determination of this point may lead to a fuller and more exact knowledge of the ætiology of the disease. This attained, an important advance may be looked for in the therapeutics of general paralysis, when recognised at an early stage.

It will be necessary for me in the first instance to sketch the appearances which I conceive to be typical in the cortex of the general paralytic, not so much for the sake of the description itself, but as affording the necessary peg on which to hang the subsequent remarks. Here I shall follow Bevan Lewis's description of the paracentral cortex. This locality is chosen because its arrangement into five layers is easily recognised in the normal brain, while the pathological changes are, on the whole, as distinct as in the frontal cortex.

Description.—To begin at the periphery, the pia mater is thickened and cellular and the epicerebral tissue is sclerosed and fibrous. The *first* layer of grey matter is thinned and contains numbers of enlarged Deiter's cells or "scavengers," which are frequently so numerous as to form a felted network with their processes. The walls of the vessels running through this layer are cellular, either through proliferation of the cell constituents of the wall or exudation of leucocytes into the perivascular space; but kinking of the vessels and cellularity are hardly so marked as in the deeper levels.

In the *second* layer of grey matter "scavenger" cells are present, though in diminishing numbers; the small polygonal nerve cells are better preserved, as a layer, than any others. Their protoplasm is granular and fatty, but the nuclei are distinct and well stained.

In the *third* layer the vessels begin to be very cellular and kinked or irregularly dilated. The ground substance is granular and cloudy, with little or no trace of nerve fibres. It has a blurred or ground-glass appearance. Scavenger cells are present especially in the neighbourhood of the vessels, though not as a rule in the numbers in which they infest the first layer. On cursory examination there may appear to be nothing but the turgid cellular vessels, scavenger cells with their processes, exuded leucocytes, and granular ground substance with a certain amount of *débris*. It is only after a more careful inspection that much of the latter is seen to be composed of remnants of smaller and larger pyramidal nerve cells. These have an irregularly spheroidal shape, and have totally, or almost entirely, lost their processes; they have a softened, half dissolved look, a very indefinite outline and a granular badly stained protoplasm. The nucleus is usually shrunken and insignificant in appearance, or else it may be swollen and distorted.

The *fourth* layer, consisting of large pyramid and ganglion cells, is greatly degenerated. It would appear that where general retrograde changes are taking place in the cortex, this layer is certain to present the most obvious and most advanced morbid characters. This is true, not only of general

paralysis, but is seen in chronic mania, in dementia, and even according to my observation in epilepsy, although here I cannot cite Bevan Lewis's authority in my support.

In the *fifth* layer of spindle-shaped or irregularly triradiate cells of a size not far removed from those of the third layer, degeneration is pronounced. It is here that scavenger cells re-appear in force, and especially where the grey passes into the white matter. Very often where these peculiar cells are scanty elsewhere, they may be fairly plentiful in these situations and even in the parts of white matter most remote from the cortex.

In the normal brain, or one slightly altered by disease, the grey matter passes rather gradually into the white. One may see what may be described as "outpost" cells extending some little distance into the white matter as solitary nerve-cell units. They are small and difficult to define from the prominence of the surrounding endothelial nuclei. These cells are very apt to suffer and disappear in general paralysis. The degenerations of the nerve cells in the fifth layer are generally those of the other layers and equal in degree to those of the *third* layer.

The cellularity of vessels in the white matter and the presence of spider cells may be mentioned, but these changes are probably of secondary importance and need not detain us here.

To the naked eye the diminution in the thickness of the cortex is plain in general paralysis. Under the microscope the diminution can be explained by the dissolution of the nerve cells and the disappearance of their processes, which implies the destruction of a vast amount of the commissural conducting fibres of which the ground substance is largely composed. The described conditions render it evident that in many instances it is hard to say where one of the different cortical layers begins and where it ends, except in the case of the first and second. Here only the demarcation remains distinct. The foregoing description may be verified easily in a typical case, or what for convenience may be considered as a typical case on the authority of Bevan Lewis and others. The variations in the microscopical appearances of different

cases are considerable, and may have helped to foster the idea that general paralysis is not a pathological or clinical entity. But the use of the term "typical" can be justified by the fact that typical cases are by no means uncommon, and that in every case, one or other of the principal histological changes will certainly be found. In most cases all will be present in a certain degree.

From a clinical point of view several kinds or varieties of general paralysis may be distinguished. In one case a maniacal onset or a well marked stage of exaltation is followed by a gradual enfeeblement of body and mind frequently accelerated by epileptoid seizures. Dissolution eventually occurs from exhaustion, caused by the latter, or from intercurrent disorder. Another may have exhibited no stage of exaltation, but a more or less rapidly advancing dementia, while in a third the mental phenomena may have been those of melancholia. One might expect from this to find at the necropsy some condition corresponding more or less constantly to the varieties and duration of the disease. As far as duration is concerned it is true that differences are found; where symptoms have been of long-standing, the brain is likely to show more general atrophy and greater accumulation of fluid both superficially and in the ventricles. But otherwise it is not found that degrees of congestion, or pachymeningitis, or adhesion of membranes have any special relation to mental symptoms, and it would be quite impossible to say what special features were displayed by the patient's disease from an examination of the cortex under the microscope.

The method of examining fresh brains microscopically which has been elaborated at the West Riding Asylum has been an enormous advantage in the study of the cortex in disease. By this method and staining with aniline blue-black are obtained results which for clearness and accuracy are not nearly approached by hardening methods. But the process presents considerable difficulties in practice. We not unfrequently find that complete sections of the cortex are hard to obtain; the peripheric portion has an unconquerable tendency to fray away, or perhaps after the sections have

been cut with apparent success, on removal from the stain they are found to have taken it up excessively and coarsely, and have a blurred or dragged appearance. This is a frequent occurrence in general paralysis, and it becomes a matter of difficulty to decide how far the condition is actually morbid and how far due to faulty sectioning. The difficulty is indicated by the fact that one section may be very much better than another, and even one part of a section may be better than another. From a considerable experience in the preparation of brain sections, not only in general paralysis, but also in epilepsy, I should be inclined to say that the blame for the unsatisfactory appearances should be equally divided. That they are "artefacts" is shewn by the occurrence of one or two well preserved sections, among possibly a dozen bad or indifferent. But it is also evident that the preponderance of the latter is due to some peculiar change in consistence of the cortex which requires a very exact degree of freezing. If this is not attained, the faulty consistence results in a dragged appearance of both cells and ground substance, as I have mentioned, and renders appearances deceptive and description useless.

Another difficulty consists in the patchy nature of the pathological changes. The morbid processes are not uniform, nor do they shade off gradually into the normal, as one passes from one region of the brain to another. Not only in adjacent convolutions, but in different parts of the same convolution and the same section we may see the most marked cell degeneration, or vascular change, or scavenger cell proliferation, side by side with a condition comparatively little removed from the normal. This makes it impossible without cutting and examining an inordinate number of sections to say with certainty that some special pathological feature is absent from the brain *in toto*. The feature that occurs to me just now is the scavenger cell which is rightly considered when present in large numbers to furnish a highly characteristic appearance in general paralysis.

A few remarks on the nature and appearance of "scavenger" cells may not be out of place. In a normal brain the cellular elements of the connective tissue comprise here and

there a corpuscle which can be distinctly seen to possess numerous fine processes—the Deiter's cells. These may be considered to furnish the normal type of the greatly enlarged and enormously active lymphatic element which from its numbers would appear to take origin, not only from cells which can be seen to possess processes, but also from those whose processes are invisible. The "scavenger" cell varies in appearance according to its stage of growth and the period of the disease. *A priori* reasoning would lead us to expect these products of a morbid activity to have distinct periods of growth, of full activity, and of decadence, and this is fully confirmed by microscopic examination of a number of cases. In describing a typical scavenger cell at the zenith of its power we should find it a protoplasmic cell of many processes. These latter vary much in length and degree of straightness, but their outlines are very distinct, and one or more of them is always attached to a vessel wall in the vicinity, though this may not appear in the section. At the point of junction can be seen a small collection of *débris* and pigment. The processes, which are doubtless hollow tubes, are more defined and take on a deeper stain with aniline blue-black than the body of the cell. The body of the cell is composed of a granular, sometimes slightly pigmented, mass of protoplasm. The shape is irregularly spheroidal and the nucleus is not always to be distinguished. This may be due to actual disappearance or merely to an excentric position. The size of the body of the cell varies considerably even at the period of greatest activity, but they may be so greatly developed as to surpass the mutilated remnants of the adjacent nerve cells, *e.g.*, the pyramid cells of the third layer. The general appearance gives the impression that the "scavenger" cells are attacking and breaking down, in fact actually battening upon the nerve elements. We shall return to discuss this point and its limitations later on.

In some cases on examining a good number of sections we may be unable to find a single adequate representative of the "scavenger" cell, though other pathological conditions may be fully developed. For instance, the disappearance of nerve cells and their processes may be quite as marked as I

have described in the typical section, and the vessels may be kinked, aneurysmal, and cellular, and yet not a scavenger cell to be seen. This strangely negative appearance raises the question of the significance and nature of the scavenger cell, and of its action here and in other morbid conditions. The origin of these elements in the Deiter's cells is not likely to be contested, nor could they well be considered as anything else than a hypertrophy of the finest lymphatic element of the normal brain. Their appearance and connection plainly betray their character, and their presence in association with broken down cell elements suggested the descriptive name of "scavenger." As a descriptive name it is a good one, and the analogy can be pushed to the full. The function of a scavenger is to remove refuse, effete and useless material. But Bevan Lewis enlarges the function of these cells still further by attributing to them an active and aggressive part in the production of the diseased condition. He figures them as attacking and disintegrating the nerve cells with their processes which he regards as suckers, not in general paralysis alone, but also in alcoholic insanity. From what I have seen and from what I have said above concerning their not infrequent absence, it would appear to me that the role of these cells is probably a secondary one. The function of their originals is doubtless to remove the effete material produced by the ordinary metabolism of the nerve tissue. Is not the stimulus of a vastly increased catabolism sufficient to account for the overgrowth of this fine lymphatic element? The function of a scavenger is to remove rubbish, not to produce it. I concede the analogy established by their title to the full, but have been unable to go further and recognise an originating and aggressive action in these cells. If their aggressive and originating action were a reality we should hardly expect to find nerve cell degeneration without their presence. We should also expect to find the scavenger cell in the greatest prominence where the degeneration is best marked. But this is not the case. The most typical nerve cell dissolution may be seen without a single scavenger cell in evidence. In fact it is, as mentioned before, usual to find a much larger number of these cells in the superficial cortical

layer where true nerve cells are wanting and at the junction of the grey and white matter than in the third and fourth layers where a more marked degradation has taken place in all the nervous elements.

It may be asked why this tendency to degeneration is exhibited in such a special degree by the cells of the third, and more particularly the fourth cortical layer. The explanation is a quasi-mechanical one. It stands to reason that the processes of nutrition in a cell are conducted with an ease or difficulty corresponding to the extent of surface exposed to the nutrient fluid as compared with the bulk of the cell contents. The smaller the cell, the larger relatively is the surface it possesses to subserve the processes of nutrition, and the more easily do the necessary anabolic and catabolic changes proceed.

Ceteris paribus, a converse statement may be made with reference to large cells. Hence, when there is a general inadequacy of, or strain on the nutrition of the nerve elements, it is the largest cells which break down the soonest. And it is possible that the size of these giant and large pyramid cells connotes a higher differentiation in development, and a corresponding diminution in reparative power. The degeneration of these elements is so common a feature in morbid brains that I fail to see any other explanation that is satisfactory. In correspondence with this view we often find in general paralysis that these large cells are much broken down and may be converted into *débris* and unrecognisable, while the smaller pyramidal cells of the third layer are easily to be discerned though, of course, much altered.

The removal of effete material is as essential to a healthy function as a proper supply of nutriment. In considering the want of relationship displayed between the degrees of nerve-cell dissolution and the presence of scavenger cells, can it be said that the latter are to blame for the degenerative processes in the nervous tissue? Is it not just as probable that this vast hypertrophy of the lymphatic element is the only method the brain has of keeping the nerve elements functionally active? The fact that this vast hypertrophy is an important sign of a diseased brain does not prove it to be

essentially noxious. In chronic renal disease or general arterial sclerosis, hypertrophy of the left ventricle is a common accompaniment. This hypertrophy is undoubtedly a departure from the normal and a valuable sign of the presence of these diseases, but its object is in reality a beneficial one. It hypertrophies merely in order to enable the circulation to be carried on efficiently in the face of a generally increased resistance to the blood current. If untoward circumstances occur such as cerebral hæmorrhage it is the ruptured vessel which is to blame. Similarly, may we not say that the probabilities are large that scavenger cell formation is a compensatory process? It is only in cases of some standing that this formation is well displayed, while again in those that have survived long, the activity of the lymphatic element diminishes and a sclerosis replaces the previously well developed network of protoplasmic cells and processes. Why the occurrence of these cells should be so uncertain both in quantity and locality is difficult to say. The doctrine of individual idiosyncrasy is a convenient refuge. Similar agents acting in different organisms produce different results and act with varying severity. This will seem rather a commonplace explanation, but it is as good as I am prepared to offer. It would not be wise to re-classify general paralysis on the ground of certain histological differences in the brain, any more than it would be on the ground of variety in the clinical features. While one dies in epileptoid convulsions at an early stage, another succumbs to exhaustion after enduring three or four years of gradual decay. In spite of all, the disease is probably as much an entity as lobar pneumonia or enteric fever. It is a symptom-complex, to use a Germanism, in which one phase or constituent may predominate. Nor can the exciting cause be relied on to decide whether we have to deal with a case of true general paralysis or not. Alcoholic insanity is a form distinct from general paralysis, but there are cases in which a condition indistinguishable from general paralysis follows on this form of brain poisoning, just as ordinary maniacs or melancholics may develop a similar condition. A history of previous syphilis or excessive sexual indulgence are doubtless predis-

posing causes, though not essential antecedents. In some, an accident or injury to the head appears to have been the starting point. Mental worry in especial is allowed to have a great, if not pre-eminent, causative action. But if all these are recognised as ætiological factors, why should we deny that other irritants may actually produce the disease? If the irritation results in symptoms like those of general paralysis and similar microscopic appearances, can we not say that the disease has been actually produced though in an unusual way? A case of hydatids of the brain occurs to me, in which the irritation spreading from the base of the brain had produced a condition diagnosed as general paralysis during life, and very similar microscopic appearances in the cortex.

This digression has interrupted a consideration of the question I desire especially to bring under notice. What is the essential and primary lesion of general paralysis? There must be some changes in the nerve tissues of the brain cortex of the general paralytic even in the early stages, while the diagnosis remains in doubt, though the patient is plainly disordered in his mind. In what I have said above are embodied some of my reasons for not accepting the scavenger cell as the origo mali. The other two appearances of paramount importance are seen in the vessels and the nerve cells. These changes go, to a great extent, hand in hand; where the vessels are most thickened and cellular the dissolution of the surrounding nerve tissue will be most evident—as a rule. But this, like most other conditions, is by no means invariable. The nerve cells in a certain part may be fairly preserved with a marked proliferation of the cells of the vessel wall, and, *mutatis mutandis*, the changes in the vessels in another case, or in another part of the same brain, may be quite insignificant while the cells and ground substance are seriously disorganised. In any case according to our present knowledge the intellectual phenomena and the motor disorder must be put down as due to failure or perversion of function in the proper nervous tissue itself. It matters not whether the abrogation is produced by something acting directly on the nerve tissue or indirectly through lesions of the apparatus that supplies its nourishment.

As Gowers[1] in a recent communication puts it, "Whatever be the original process" (*i.e.*, in nervous disease) "the symptoms themselves invariably depend on minute changes. If the primary morbid process is a massive one, as a tumour of the brain, the symptoms which manifest it presence and declare its character are of a minute nature." . . . . "The effects of a gross specific disease such as an artery causing softening may differ very little from those of a degenerative specific disease."

If the disease, then, of body and mind is accompanied by a degeneration of nerve cells which can always be recognised, whether the peculiar vascular and lymphatic conditions are in evidence or not, does it not justify the presumption that the essential and primary lesion is one of the nerve cells and their processes? It is quite possible that the same cause which produces the break-down of the nerve cells and their inter-communicating fibres causes simultaneously the cellular proliferation of the vessel walls. It may be that the vascular and lymphatic conditions when well shown are the most *characteristic* appearances in general paralysis. That does not necessarily imply that they are the essential and primary factors at fault. Scavenger cells are seen in other conditions; in alcoholic insanity, in senility, to a certain extent in epilepsy and in acute meningitis. The appearance of thickened, proliferated and cellular vessel walls is not so common a feature and as far as I know is not met with in a well marked state in any other condition of brain disease. But as this feature is not so constant as the dissolution of the nervous tissues to which ultimately the symptoms of the disease must be referred, I repeat that the probability is in favour of a purely nervous origin.

What the actual cause may be we are not in a position to say as yet. It may be that it is some organic poison which under certain conditions has the power of attacking and a special affinity for the higher nervous tissues. Not all that have led a fast life and "burnt the candle at both ends," or undergone great mental strain and worry, become general

[1] Address on Neurology and Therapeutics, *British Medical Journal*, April 22, 1893.

paralytics. It is no more than a small percentage that does so, although they may suffer in other ways. Though alcohol has an affinity for nervous structures, it is only a small proportion of the intemperate that undergo peripheral neuritis. The mousy smell in general paralytics, not only of the surface but of the whole body, would seem to lend some support to the theory of a quasi-septic intoxication being the originating evil. Besides, we have an analogy in influenza. In one or two cases which have come under my notice, with acute dementia as a sequela, the softening of the brain cells was remarkable, and appearances pointed to their rapid dissolution. The vessels did not present any notable feature beyond a light yellow staining, due in all probability to the solution of blood pigment. Scavenger cells were not present. The conclusions, then, may be shortly summed up as follows :

(1) In brain disease perverted function, whether mental or physical, must be attributed immediately to nerve-cell degeneration.

(2) Nerve cell degeneration may be primary or secondary.

(3) In general paralysis nerve-cell dissolution frequently occurs without the special morbid appearances usually considered as characteristic and in a degree causative.

(4) The balance of evidence is therefore in favour of a primary nerve-cell lesion being the essential morbid condition in this disease.

SYPHILIS OF THE SPINAL CORD.[1]

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THE *post hoc propter hoc* argument has been used so freely with regard to syphilitic diseases of the central nervous system that it seems eminently desirable to be able to make the diagnosis of cerebral or spinal syphilis without reference to the etiological factor. We should be able to recognise the symptoms of syphilitic disease whether or not the patient concedes, or we can prove previous syphilitic infection. My own experience during the past few years leads me to believe that this can be done with tolerable certainty in the case of the diseases under discussion.

Some time ago in a paper on "Multiple cerebro-spinal syphilis,"[2] I referred to the morbid lesions and to the important points of differential diagnosis for cases of syphilitic disease involving both the brain and the spinal cord. In this paper I propose to confine myself entirely to the diagnosis of spinal cord syphilis.

A fresh impetus has been given to the writings on this subject by Erb's short article[3] which appeared in the *Neurologisches Centralblatt* for March 15, 1892. With the same keen clinical insight which led to admirable results in his studies on muscular atrophies and of Thomsen's disease, Erb has sought to establish "a type" of spinal cord disease which he proposes to call "syphilitic spinal paralysis."

[1] Read before the New York Neurological Society, April, 1893.

[2] *New York Medical Journal*, 1891.

[3] A detailed analysis of Erb's cases will be found in an interesting article by Dr. S. Kuh, of Chicago, published in the *Zeitschrift für Nervenheilkunde*, Bd. iii.

This special type is to be recognised by the following characteristics:

(1.) The usual symptoms of spastic paraplegia:
The peculiar gait, carriage and movements.

(2.) Marked exaggeration of the deep reflexes.

(3.) Muscular contractures slight as compared with the exaggeration of the reflexes.

(4.) Involvement of the bladder.

(5.) A slight, yet distinct disturbance of sensation.

(6.) Gradual onset of the disease.

(7.) A decided tendency to improvement.

We may concede at once that these points are well taken and that the symptoms stated above leave little doubt of the syphilitic nature of a given case. It is due to Rumpf to say that, on p. 351 of his well known monograph, he alludes to all these diagnostic symptoms in a case which was proved to be due to specific disease of the blood vessels in the spinal cord. He mentions spastic symptoms, paresis followed by paralysis of the sphincter of the bladder with incontinence of urine, exaggerated reflexes and contractures; later on in the course of the disease the diminution of sensation. The onset, too, was a gradual one. Rumpf, naturally enough, in speaking of his case which ended on the *post-mortem* table, does not refer to a tendency to improvement, and he does not insist, as Erb does, on the relatively slight muscular contractures as compared with the great exaggeration of the reflexes.

In developing his spinal type Erb recognises Rumpf's services in this field. The former may claim the special merit of attaching greater importance than Rumpf did to this difference between the intensity of development of muscular contractures and of increased reflexes.

The questions I have been trying to solve for some time past are—first, whether this Rumpf-Erb type represents the most frequent form of syphilitic spinal cord disease; and secondly, whether the cases that conform to this type represent *a* syphilitic spinal paralysis, or *the* syphilitic spinal paralysis, as Erb would have it. This is not a mere quibble of words. Much mischief was done, as every one will acknowledge, by Strümpell's procedure in describing the

clinical features, of what he supposed to be polio-encephalitis, and in labelling these cases "*Die* cerebrale Kinderlaehmung," whereas it is now well established that if his cases exist at all they constitute a very small proportion of all the infantile cerebral palsies. I do not fear a similar mistake with regard to Erb's type of spinal cord disease, but there is a distinct tendency even now among more recent writers to imply that every case of syphilitic spinal cord disease should conform in most respects to the type described by Erb.[1]

This leads to the further question: Are the symptoms described by Erb so characteristic of spinal syphilis that we should trust to them entirely, or are there other symptoms which point with much greater force to the syphilitic nature of cord disease?

For the present the question is a purely clinical one, and one of great practical importance at that. The question in pathology can be brought up after the clinical question has been settled.

Let me record in condensed form a few cases, taken from private practice, of undoubted syphilitic disease of the spinal cord, from which we shall be able to draw the necessary inferences.

Case 1.—Mr. C., age 50, was referred to me by Dr. Gibney, September, 1891, and has been under my treatment ever since.

There was a distinct history of syphilis acquired two years previously, followed by constitutional symptoms.[2] He has been married 23 years; wife has had no children. He has been an active brain worker for many years, but moderate in his mode of life. In November, 1890, he had an attack of illness, during which his right eye-lid drooped; his left leg was paralyzed. In the course of a few weeks he recovered sufficiently to get about again. He also had "*eczema ani*," and shortly after this was treated by various physicians for the tertiary symptoms of syphilis.

In the spring of 1891 he had a severe attack of influenza. A few months later in May, 1891, he developed a right hemiplegia, and a paresis of bladder and rectum. The left leg was still

[1] It is evident from Dr. Kuh's article (which has appeared since the above was written) that Professor Erb recognises mixed forms, &c., of syphilitic disease of the spinal cord.

[2] I am indebted to Dr. J. P. Tuttle for the early history of the case.

weak from the first attack. He recovered in a few weeks and was once more active in his business.

Two weeks before I first saw him he was compelled to go to bed again, his legs having grown weak and his bladder and bowels not acting voluntarily.

The condition I found him in was a rather pitiable one. In addition to complete spastic paralysis of both lower extremities, there was complete paralysis of both upper extremities; there was no increase of the wrist and elbow reflexes, but a decided tendency to atrophy of the interossei particularly in the left hand. The knee jerks were enormously exaggerated; there were *extreme contractures* of the adductors of the thighs, and the very marked contractures of the posterior leg muscles inhibited the ankle clonus. The pupils were normal, speech normal; sensation in both lower and upper extremities was normal at the first examination. The electrical reaction of all nerves and muscles was entirely normal. There was retention of urine and inability to evacuate the bowels. This group of symptoms has been materially modified from time to time.

The involvement of upper and lower extremities with symptoms pointing to a very irregular invasion of the various systems of the spinal cord left no doubt in my mind as to the nature of the trouble, and in spite of very discouraging circumstances, I adhered to, and pushed, the mercurial and iodide treatment[1] to the extreme. During the first week the symptoms increased in severity. The paralysis became absolute and anæsthesiæ were soon associated with the paralysis. The anæsthesia was evident only in both feet and legs up to the middle of the thighs and over the sacrum and lower dorsal spine. There was no anæsthesia in the upper extremities, but very decided atrophy of the interossei. Within two weeks of the beginning of the treatment, while the conditions of the lower extremities remained unchanged, the upper extremities recovered completely and have been entirely normal ever since. Scarcely had these symptoms been recovered from before the intensity of the process in the lower cord seemed to increase. A bed-sore began to form which in spite of the most careful nursing increased in size until it was large enough to put one's fist in, and for fully eight months it was a threatening feature of the case, but finally healed completely under the surgical skill of Dr. Gerster. There has been

[1] Inunctions of six grammes of mercurial ointment daily, and gradually increasing doses of the saturated solution of the Iodide of Sodium, from 10-150 drops, three times daily.

a gradual improvement in the paralysis, so that after much patient treatment he is now able to walk about with the assistance of his crutch and an attendant, with every prospect of regaining the normal use of his legs unless a sudden relapse should set in. All anæsthesiæ have completely disappeared, the contractures are relaxed (the ankle clonus is now apparent), but spasmodic twitchings of the legs are still quite frequent. The patient has also recovered from several attacks of cystitis, but is still suffering from a specific ulcer of the rectum.

Recovery in this very severe form of spinal syphilis I consider fortunate enough, and doubt if it would have been possible if the symptoms had not been due to a specific process.

Please note that the contractures were extreme and that at one period very marked sensory disturbances existed.

Case 2 resembles the first in many particulars and still has run a very different course.

Mrs. X., a lady age 53, having five children, all healthy and married, was first seen in June, 1891. She was in good health until two years previously, and extremely active in spite of her weight of nearly 250 pounds. After the "blizzard," March, 1888, she had a troublesome cough and for a long time did not feel well. In spring of 1889 began to notice a heaviness in the left leg; this would remit at times and then would grow worse again, so that she was accused of having an hysterical affection. The trouble was restricted to the left leg for nearly two years, before the right leg became similarly affected. A few weeks before I saw her she was sent to Atlantic City by a physician who thought that the change of air would have a favourable influence upon her supposed functional paralysis.

The first examination revealed a very different state of things: (*a*) Extreme and absolute spastic paraplegia; not a muscle of a toe even could be moved. (*b*) Extreme contractures, so that the knees would be firmly clapsed together unless forcibly separated by pillows. (*c*) Violent spasmodic twitchings which gave rise to excessive pain. (*d*) There was complete anæsthesia of both legs as high up as a line passing two inches above the symphysis pubis. (*e*) Loss of faradic response without alteration of galvanic formula in all leg muscles and nerves. (*f*) No tendency to bedsores; and (*g*) absolutely no trouble with bladder or rectum.

The upper extremities were not affected. No history of traumatism or of unusual exposure, and, I beg you to remember, no history of an acute infectious disease. In the absence of these predisposing conditions—and more particularly on account of the

extreme spastic symptoms associated with disturbances of sensation and with changes in electrical reactions (atrophy could not be made out on account of adispose tissue) without, however any interference with vesical and rectal reflexes, and no tendency to formation of bed-sores, I made the diagnosis of a specific myelitis. No other pathological process is as selective as this one. The symptoms pointed to involvement of the lateral columns, of the posterior roots or posterior gray matter, and also of the anterior horns or roots on both sides, and yet the column of Clark and the central grey matter were seemingly not affected.

I have persisted in specific treatment up to the point of tolerance with gratifying results, although the proof of any specific illness was a long time forthcoming. Some nine months after the beginning of treatment the lady's husband consulted me for very persistent headaches. Suspecting the nature of the trouble, I discovered not only that he had acquired specific disease many years since, but that he had been under a distinguished surgeon's care for bone syphilis only a year ago. The further progress of the wife's case has been marked by a gradual restoration of the electrical reactions, by total disappearance of the anæsthesiæ, by the cessation of all contractures and of the spasmodic twitchings, and by the slow and steady diminution of the spastic paralysis. She has regained moderate use of all the muscles of the lower extremities, but is still confined to her chair, as her legs will not yet bear her tremendous weight; but I expect her in a few months to learn to walk, as single efforts to stand have been successful, and she moves legs and feet very readily while sitting in a chair. I have not seen any but specific cases of spinal cord running such a chronic course and yet showing a tendency to improvement after the lapse of one or two years and even longer. I hold that in this case, next to the very remarkable improvement, the entire absence of bladder and trophic symptoms compared with the intensity in development of the other symptoms is the most characteristic diagnostic feature.

I could parallel this case by at least two others, but I do not wish to go into wearying details.

Case 3 is that of a colleague now practising in Hot Springs, Ark. He is 36 years of age; married, no children; formerly in good health; first symptoms of fatigue on slight exercise in 1889; began to look pale and run down. Was in Ottawa, Canada, at the time, and went to sea for a couple of years; did not improve, and he began to have rheumatic pains about joints, and suffered somewhat from dizziness. After his return he developed a very

marked spastic gait which has slowly increased up to the present time; a slight irritability of the bladder, and loss of sexual power were symptoms from which he suffered at various times; but both these functions are now normal. He suffers most, however, from headaches. On further questioning I learned that one year ago after the onset of spastic symptoms he had some difficulty in speaking, associated with a numbness of the left side of the body; once had an attack of aphasia lasting 24 hours, and remembers a temporary weakness of left hand. As a result of my examination, I noted inequality of pupils, no other head or ocular symptoms; grasp excellent in both hands, but very exaggerated wrist and elbow reflexes; spastic gait with considerable rigidity, so that in walking the knees are pressed quite closely together; exaggerated knee jerks; no anæsthesia anywhere, no trophic symptoms, no tremors, no stammering speech, &c. I made the diagnosis of spinal syphilis, and then learned that fifteen years ago he had been inoculated with syphilis on the middle finger of left hand. I urged repeated mercurial and iodide treatment, but this has not relieved the spastic symptoms.

Please note again extreme spastic symptoms with contractures, slight involvement of the bladder, no sensory symptoms, and no atrophic symptoms, proving once more the selective character of the process. The case resembles in most respects those described by Erb as typical of syphilitic spinal paralysis.

Case 4.—This case, differing as it does from all other cases of spinal or cerebro-spinal syphilis I had seen, led me to seek for safe guides in differential diagnosis. I saw the young man, F. I., age 26, in consultation with Dr. Lustgarten, Oct. 14, 1892. He comes of a very markedly neurotic stock with the history of insanity in the family, and has one sister who is deaf and dumb.

Four years ago he had acquired undoubted syphilis. Since last spring has experienced difficulty in walking and in the use of his hands.

There was also the history and evidence of paralysis of the right superior oblique. At the first examination the young man was quite unable to lift himself up in bed; there was distinct atrophic paralysis of both trapezii, of infra and supra spinati, of deltoids and pectorals; a paresis of the entire arms and hands; grasp being almost nil.[1] All forms of sensation, particularly tactile and temperature sense, were normal. This was true of the upper extremities and of the lower extremities and on the trunk to the level of the umbilicus. Elbow reflexes could not be

[1] At this first visit a careful electrical examination could not be made.

elicited. The lower extremities were much emaciated, more so the right than left; great weakness, scarcely able to lift legs an inch from the bed, and yet if put on his legs could stand a little; no ataxia, no absolute paralysis anywhere. Right knee jerk lost, left knee jerk very feeble; all superficial reflexes lost; no disturbances of vesical and rectal reflexes: no bed-sores, but a severe burn in the anæsthetic area on the right side just below the margin of the ribs.

The diagnosis of wide-spread syphilis was clear to me, although the paralysis tended to the atrophic rather than to the spastic type.

The good results of treatment were very evident at my next examination about two months later, Dec. 6. Patient was able to sit up in bed and to walk about though feebly; the pupils now reacted to light, though sluggishly, and the muscles about the shoulder joint had recovered surprisingly from the atrophic condition. Normal sensation had returned, leaving a relatively small area of anæsthesia on the trunk. It is worth noting that at a still later examination the area of anæsthesia had again increased, while all other symptoms had receded.[1] Grasp of hands nearly normal, all muscles with the exception of the extensor groups in both forearms having been restored to their normal volume. Faradic responses diminished but present in all muscles even in the extensors, with the exception of the anterior tibial group of the right side. Both knee jerks can now be elicited by reinforcement, and his mental condition has much improved.

Four months after first examination patient was able to call upon me at my office, improved in every respect, walking without assistance and simply using a walking-stick to get about with. He was still affected with double vision, in consequence of paralysis of right superior oblique.

If we except the slight ocular trouble, this was a spinal case clearly enough. The paralysis was of the atrophic and not of the spastic order. The involvement of both the upper and the lower extremities; the unequal development of the symptoms (the paralysis and anæsthesia being very pronounced, while the bladder was not involved, and the electrical conditions and the reflexes were less distinctly altered, and trophic disturbances were wanting entirely), this peculiar and unequal development of symptoms, as well as the rapid recovery, proved the case to be one of spinal syphilis with slight cerebral involvement.

[1] Drawings illustrating this variation in the areas of anæsthesia were exhibited at the meeting.

In the cases just recorded the diagnosis of spinal cord syphilis can hardly be questioned. I share the fate of other writers, in not being able to offer *post-mortem* evidence of these diagnoses. Recent syphilis of the central nervous system if properly recognised should not lead to *post-mortem* examinations, excepting in rare instances; and it is true, moreover, of all syphilitic diseases that the clinical manifestations are in many respects far more reliable than are the results of histological examinations of the diseased tissues, for, curiously enough, the pathologist in making his diagnosis has to rely very largely upon points obtained by clinical examination.

Now what were the features which led to the diagnosis of syphilis in the cases I have detailed? In one there was wide-spread atrophic paralysis, in three of them there was spastic paraplegia of the most pronounced type. In these the reflexes were excessively exaggerated. In two of them the muscular contractures were slight; in one of them they were extreme. In one case there was permanent involvement of the bladder. In the others no involvement. In all but one of them sensation was disturbed. In two the onset was gradual. In the others it was quite sudden. All of them have shown a decided tendency to improvement.

I have mentioned first the features in which they conformed to Erb's type, but in other and equally important, if not more important, points they differ from that type. In one instance there was a distinct atrophic paralysis, with all the symptoms that pointed to a wide-spread affection of the gray matter of the cord, that would have reminded one of a poliomyelitis, with the exception that, as predicted on the strength of the syphilitic character of the disease, the patient made a very rapid recovery.

In Case 1, in which the diagnosis of syphilitic disease is to my mind beyond dispute, the contractures were extreme: the bladder has remained involved up to the present time, and bed-sores formed as in the most typical case of acute transverse myelitis.

These points of difference did not conceal the true nature of these cases, and I beg of you now to consider a few facts which have impressed themselves upon my mind as more

characteristic of spinal cord syphilis than any other I have been able to fasten upon.

(1) The most striking of all is the unusual distribution of the disease over the greater portion of the cord, involving as it did in several cases the cervical, dorsal and the lumbar enlargements.

(2) The relatively slight *intensity* of the morbid process as compared with the *extensive area* involved, as evidenced by the preservation of some of the functions of the cord with complete loss of others.

(3) A rapid dwindling of some of the symptoms, and the very chronic persistence of others. In several of the cases, for instance, the anæsthesia lasted but a very short time, while the paralysis was recovered from with extreme slowness.

(4) The very frequent history of other symptoms pointing to specific disease in the same or other parts of the central nervous system.

I shall be inclined to suspect specific disease of the cord if the patient presents symptoms of paralysis, whether they be of the spastic or flaccid character, and whether the contractures be slight or not, provided he furnish evidence of a morbid process affecting a very large part of the cord, and yet showing a relatively slight intensity at any given level of the cord; if, for instance, he exhibit the symptoms of extreme paralysis, spastic or atrophic, with partial or slight anæsthesia of the parts paralyzed, with little or no involvement of the bladder, and particularly if we can find traces of specific disease in other parts of the central nervous system.

These cases remind one more or less of those which were formerly diagnosticated as irregular cases of subacute or chronic myelitis; and if we refer for a moment to the nature of the morbid process we can readily understand why this irregularity in the development of the symptoms should be the most characteristic of all of the diagnostic features of these cases. There is not in them as in the cases of acute myelitis a morbid process which is rapidly destructive and which rapidly advances across the entire cross section of the

cord, involving all the symptoms due to loss of function of the various spinal systems. If the syphilitic disease be the result of a specific endarteritis of the vessels of the cords, we know that some and by no means all of these vessels are affected, and that the disease advances slowly from one group of vessels to another. If there be diffuse specific infiltration it also invades but very slowly one part after the other. It has a remarkable tendency, too, to increase for a time and then to recede, whether as a result of treatment or not, and then possibly to increase with renewed force. If the infiltration start from the meninges it invades most frequently the lateral columns first, often at symmetrical points, and advances very slowly from white to gray matter. The intensity of the process is spent often upon the lateral columns, hence the frequency of the spastic symptoms. It may invade the gray matter, giving rise to sensory symptoms, sometimes to atrophic symptoms. It is very apt to recede from the gray matter, restoring sensation, the conditions of nutrition and the normal condition of the bladder; but it seems to halt at the lateral columns, leaving the spastic symptoms intact for a very long time.[1]

If we try to harmonize the clinical symptoms with the pathological conditions it would seem much more natural to lay greater stress upon those symptoms which are due to the peculiar retrograde features of the morbid process, than upon those symptoms which are dependent upon the area attacked by the disease.

For all these reasons it seems better to fasten upon *the relatively slight intensity of the disease*, and consequently of all symptoms, *as compared with the extensive area involved*, and upon the *tendency to remissions and relapses* than upon any mere association of symptoms due to involvement of the different parts of the cord at any one given area. I do not mean to dispute the existence and the propriety of establishing Erb's type of spinal disease, but I do believe that if we do not regard other and broader points of diagnosis we shall often fail to recognise spinal cord syphilis.

[1] In a paper entitled "Tabes and Syphilis" (not yet published), read before the American Neurological Association, July, 1893, I have laid particular stress upon the anatomical features of this process.

ON DIABETIC NEURITIS, WITH A CLINICAL AND PATHOLOGICAL DESCRIPTION OF THREE CASES OF DIABETIC PSEUDO-TABES.

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SINCE Marinian and Bouchard (1) first pointed out, in the year 1884, the fact that the knee jerks were frequently absent in cases of diabetes mellitus, the attention of medical observers has been especially drawn to the various nerve troubles and complications which not uncommonly arise during the course of this malady.

Buzzard in 1886 attributed the loss of the knee jerks in some cases to peripheral neuritis. Negative pathological evidence in support of this view was also obtained through the researches of Rosentein (2), Niviere and others. In the cases reported by these observers, the spinal cord when microscopically examined was found to be healthy.

Clinically Leyden (3) has divided the neuritis of diabetics into three groups, *i.e.* :—

(1) The hyperæsthetic or neuralgic form, in which there is more or less severe pain. V. Ziemssen first ascribed the various neuralgias which not uncommonly occur in diabetes to be the result of neuritis. These neuralgias are not infrequently symmetrical in their grouping.

(2) The motor or paralytic form. In this variety there is paralysis of varying degree of the muscles of the legs, or of single muscles, or groups of muscles.

(3) The ataxic form—pseudo-tabes. Here there is ataxia, and various sensory symptoms, such as numbness, formication of the feet, &c. The knee jerks are lost.

Whilst agreeing with Leyden that cases of diabetic neuritis may to a large extent be divided into these three different groups, I believe the clinical picture would be truer and more complete if we restricted them to two fairly distinct varieties, *i.e.*—

(*a*) The motor or paralytic form.

(*b*) The sensory or ataxic form.

In this latter variety I would include Leyden's hyperæsthetic or neuralgic group, believing that the symptoms characteristic of this class do in many instances develop into more serious nerve complications. In this group the sensory, vaso-motor and trophic nerve fibres are chiefly affected, and the clinical symptoms presented bear a very close resemblance to those of locomotor ataxy.

In the *British Medical Journal* of March, 1887, I drew attention to those cases of diabetes in which there was not only loss of the knee jerks, but also a certain degree of ataxia, and ascribed the symptoms to a somewhat widespread peripheral neuritis. To this type of case the term diabetic ataxia, or diabetic pseudo-tabes, may I think be very suitably applied. In the same year Dr. Pavy (4) in his address on diabetes at the Washington International Medical Congress described a similar class of case.

"He stated that this form of ataxy occurs in persons past the middle period of life, that they complain of pains of varying character in the limbs, and that the ataxy is not exactly the pure form met with in tabes dorsalis. For the most part these persons can stand and maintain their balance fairly well with the eyes shut, but they walk with difficulty. They cannot properly maintain their balance. Associated with these symptoms there is some anæsthesia and hyperæsthesia, and various forms of paræsthesia. They often feel as if they were walking on pebbles. The disease is most marked in the lower extremities; sometimes, however, the upper extremities are also affected. The patient complains of an aching of the bones at night. The condition appears to be due to peripheral neuritis."

To this vivid description of the symptoms presented by cases of diabetic ataxia I would only add that the knee jerks

are diminished or frequently absent, and that these patients are occasionally liable to vaso-motor and trophic troubles, as evidenced by œdema, erythema, and perforating ulcers of the feet.

Death may also result from gangrene of the extremities. There are no doubt cases of diabetic neuritis which present motor and sensory symptoms; but in the main I think their division into motor and sensory varieties is a correct one.

Some twenty cases of diabetic neuritis have been recorded during the last few years, notably those by Leyden, Charcot (5), Buzzard (6), Althaus (7), Auché (8), and Eichhorst (9). They have varied much in their symptoms; in some the motor and in others the sensory nerves have been chiefly affected. As our knowledge of the disease increases I opine that it will be found that the sensory nerve fibres are especially liable to be affected in diabetes.

Although it will be seen from what has already been stated, that clinically diabetic neuritis has been abundantly recognised, very little pathological proof of this lesion has been forthcoming. Indeed, with the exception of a case published by myself (10) in the *Lancet* of July, 1887 (an abstract of which follows), and of the observations of Auché in 1890, and Eichhorst in 1892, I have been quite unable to find distinct pathological evidence of this form of neuritis.

I trust that the cases appended will add still further to our pathological and clinical knowledge of this disease. They belong to the sensory or ataxic group, and may very properly be termed cases of diabetic pseudo-tabes.

Case 1.—A. D., aged 56. Diabetes. Ataxic symptoms, perforating ulcers of both feet; coma; death.

Frame work knitter and hawker; was first seen on May 27th, 1886. For eighteen months past he had passed large quantities of urine, and had suffered from great thirst. He was never of temperate habits, and was addicted to "drinking bouts."

No history of gonorrhœa or syphilis could be elicited, but strong presumptive evidence of venereal taint was present in the form of two scars, one in each groin, and various suspicious-looking marks on both legs. Eight months previous to admission he complained of pains in the legs and stated that his feet felt dead.

At Christmas, 1885, he caught cold and suffered from an aggravation of the foot symptoms, together with abdominal pain and vomiting. He was described at this time as walking on his heels. In February, 1886—three months and a half before his death—the ulcers of the feet first appeared.

When first seen by me the patient was suffering from bronchitis and symptoms of commencing diabetic coma. When supported he could, however, manage to walk across the room. His gait was distinctly ataxic. The arteries of the upper limbs were evidently diseased. The pupils reacted feebly to light. There was diminished cutaneous sensibility of both feet, and the lower thirds of both legs. The knee-jerks were totally abolished. The skin over the legs and feet was of a livid hue, and below the normal in temperature. A fœtid odour emanated from both feet. The ulcers commenced as corns. The largest ulcer, of the size of a two shilling piece, was situated over the plantar aspect of the meta-tarso-phalangeal joint of the right great toe. An ulcer of the same character but of smaller size was situated over the plantar aspect of the head of the fifth metatarsal bone of the opposite foot. Carious bone could be felt in each case. The patient's urine was acid; sp. gr. 1030, contained sugar and a trace of albumen.

He died of diabetic coma on the fourth day after coming under my observation. There was no history of gout, rheumatism or injury in this case.

The spinal cord, and the nerves of the right lower limb were examined. The naked eye changes which were detected were (1) considerable enlargement of the lower third of the posterior tibial nerve. The nerve was surrounded by inflammatory material, in which was embedded the atheromatous posterior tibial artery; (2) the plantar terminations of the nerve were discoloured and diminished in size.

The cord and peripheral nerves were examined by Mr. Bowlby of St. Bartholomew's Hospital, who demonstrated the following changes. In the lumbar region of the cord there was marked atrophy of the cells of the grey matter, which were granular and had lost their processes. In the dorsal region the changes were even more marked. There was no cell exudation in either the white or the grey matter. In the peripheral nerves the changes were most evident in the posterior tibial trunk, where the white substance of Schwann was replaced by granular material which did not stain black with osmic acid. Some of the axis cylinders were normal, but many were atrophied and the subject of fatty

degeneration. The anterior tibial nerve showed a similar degeneration, but in a lesser degree, and in the sciatic the bulk of the fibres were normal. There was also a slight increase of the connective tissue basis.

Dr. Byrom Bramwell of Edinburgh also described similar changes in the peripheral nerves of this case, together with well marked vascular disease involving not only the larger vessels, but spreading also to their minute subdivisions. There was no ataxic lesion in the spinal cord.

Case 2.—Charles B., aged 72. Diabetes. Ataxic symptoms; gangrene; death.

This case came under treatment four years before death took place. For ten years previous to this date he had suffered from double sciatica, and for some years pain of a lightning and burning character had been especially marked in both lower extremities.

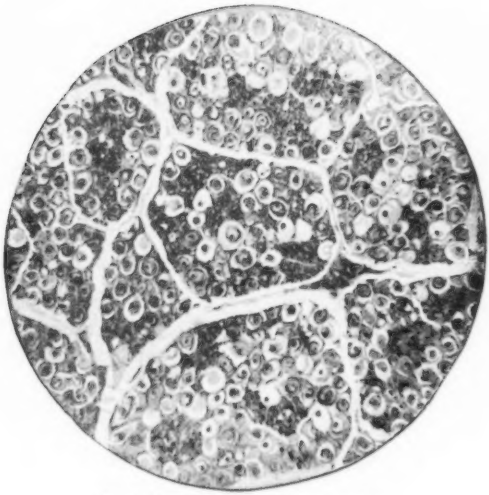
When first seen he complained greatly of the above-mentioned pain, its seat of greatest intensity being in the left foot and leg. Both knee-jerks were diminished, and could only be obtained on reinforcement. There was marked muscular inco-ordination in the dark or with closed eyes, but no evidence of paralysis.

Anæsthesia was present to a varying extent in the toes of both feet. To a lesser degree this anæsthetic condition was also present in the feet and lower thirds of both legs. He frequently complained of burning and pricking sensations; most of the teeth had fallen out in a fairly sound condition. The pupils reacted to light and accommodation.

He suffered from great thirst and passed large quantities of urine, which was acid, sp. gr. 1025, and contained sugar. There was no history of alcoholism, syphilis or rheumatism.

This patient occasionally suffered from a peculiar erythematous and œdematous condition of the feet, which appeared to be distinctly co-incident with an increase in the amount of sugar passed in the urine. During one of these attacks of "erythematous œdema" a small ulcer formed on the plantar aspect of the left great toe. This ulcer was surrounded by a very marked anæsthetic zone. The urine during this attack increased in quantity, sp. gr. 1030, and contained much sugar.

The course of the malady in this case was varied, but pain was always a constant and distressing symptom. For some time before death the loss of the knee-jerks was complete. He died of gangrene of the left foot and lower third of the left leg in July, 1891.



Healthy Nerve. Weigert stain.

C. B. 72. Sciatic, showing increase of connective tissue basis. Weigert stain.

The gangrene commenced in the ball of the left great toe (the site of the previous ulcer), and gradually extended upwards. It was of two months' duration. The anterior tibial, posterior tibial and sciatic nerves all showed a degeneration very similar to that described in the first case. These changes were most marked in the posterior tibial trunk. There were most distinct arterial changes, consisting of atheroma of the large artery, and thickening of the coats, and in some instances of blocking up of the smaller microscopic blood vessels.

I am much indebted to Dr. W. B. Ransom for the microscopic examination of the nerves in this and the following case.

Case 3.—William A., aged 62. Diabetes, ataxic symptoms, gangrene, death.

This case was under observation for a period of nearly four years, October, 1887, to May, 1891. When first seen he gave a history of having suffered from great thirst for more than two years. During this time he had also passed an excessive amount of urine. He complained of shooting and burning pains in both lower limbs and feet, but more especially in the right foot. Cutaneous sensibility was diminished in both feet.[1] Both feet were extremely painful, and presented the condition of "erythematous œdema" already described in the foregoing case.

There was slight muscular inco-ordination; no paralysis. The knee-jerks were diminished but not totally abolished; the pupils reacted to light and accommodation; urine, sp. gr. 1036 and contained sugar but no albumen or peptone.

The symptoms in this case (although varying from time to time) gradually became aggravated until about four weeks prior to his death gangrene commenced in the right great toe and extended up the foot to the junction of the lower with the middle thirds of the right leg. Mortification also set in, in the left toe and foot, but did not extend to the leg. Death was due to exhaustion. For some months before death the knee-jerks could not be obtained.

The sciatic, anterior and posterior tibial nerves were examined and showed marked parenchymatous degeneration, the changes being very similar to those found in the nerves of the foregoing cases. Vascular disease was also present in the form of atheroma of the larger arteries, and of thickening of the walls and blocking up of the lumen of the smaller blood-vessels. There was no history of alcohol, syphilis or rheumatism in this case.

[1] This refers to the condition of the feet generally, but not during an attack of erythematous œdema.

Clinically the chief points to be noted in the foregoing cases are :—

(1) Their chronic nature.

(2) The presence of distinct ataxy.

(3) The absence of any definite paralysis.

(4) The age of the patients, all the cases occurring at an age past the middle period of life.

(5) The preponderance of sensory, vaso-motor, and trophic symptoms, and

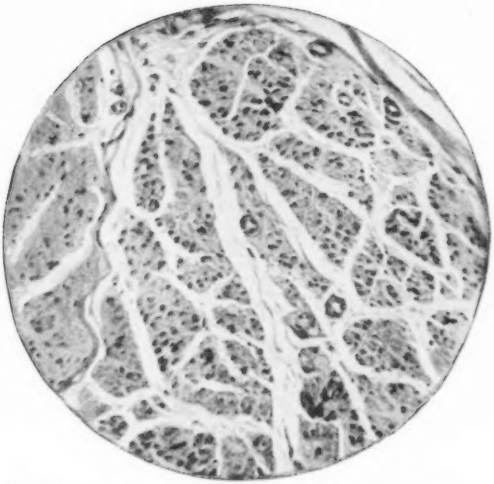
(6) The association with the nerve-symptoms of well-marked arterial changes.

Dr. Byrom Bramwell has pointed out, that in cases of peripheral neuritis, where the sensory nerve fibres are chiefly affected, the course of the disease is usually slow, and the prospect of cure remote, whereas the reverse is the case when the motor nerve fibres are mainly involved. These cases certainly lend weight to this dictum.

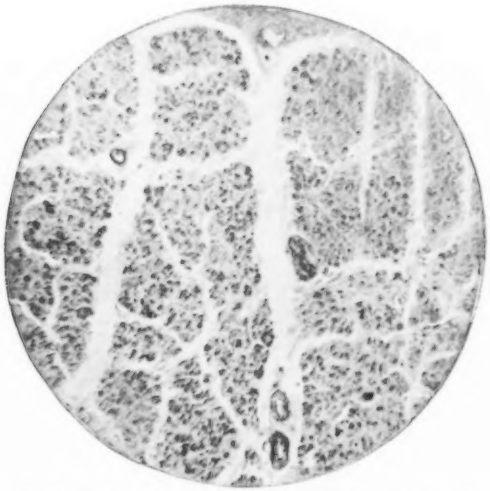
The presence of definite ataxia in peripheral neuritis pure and simple has been denied by the late Dr. Ross, of Manchester. It appears to me that this statement cannot be altogether accepted as a true one. In none of the patients was there any distinct paralysis, and in all there was more or less definitely marked ataxia. In one case there was no tabetic lesion found in the cord. Here one microscopist describes atrophy in the cells of the grey matter. No mention is made by the other examiner of this lesion.

It is therefore, perhaps, an open question, whether these changes were *ante-* or *post-mortem*. Examination of the spinal cord was not allowed in the other cases. No relationship could be established between the severity of the nerve symptoms and the amount of sugar in the urine, if we except the condition of "erythematous œdema," which appeared to be distinctly coincident with an increase in the amount of sugar passed.

The etiology of diabetic neuritis is manifold. We shall not discover it to be solely resident in some specific toxic poison. It is quite possible—nay more, probable—that some of the acuter forms of the malady occurring in young people may be thus explained; but I consider such a theory



W. A. 62. Post. Tibial Nerve. Hæmatoxylin stain. Increase of nuclei, &c.



C. B. L. Post. Tibial Nerve. Weigert stain. Degeneration of Nerve Fibres. Increase of connective tissue, &c.

insufficient to explain all the clinical and pathological facts of cases such as the above.

Here we have other factors at work. In the first place there has been long continued malnutrition, and in the second there is found extensive vascular disease.

Whilst I do not deny that a specific toxic poison may not have had a share in the causation of the neuritis in these cases, I would at the same time ascribe the disease mainly to vascular disease and malnutrition. Vascular changes were apparently as much a part of the morbid process as peripheral nerve degeneration.

The posterior tibial artery is perhaps the one most liable of all the arteries of the body to atheromatous degeneration, and it is in the posterior tibial nerve trunk supplied by branches of this vessel that the nerve degeneration was especially marked.

The condition of erythematous œdema is apparently very closely allied to the vaso-motor neurosis described by Weir Mitchell under the name of erythromelalgia.

The treatment of these cases resolved itself into the treatment of diabetes and the maintenance of the general health.

Morphia and antipyrine were of great service in allaying the constant pain. Operative interference would, I believe, only have acclerated death.

BIBLIOGRAPHY.

(1) (*a*) MARINIAN. "Contribuzione-allo-studio clinico dei riflessi tendinei," *Rivista Clinica di Bologna*, 1884.
(*b*) BOUCHARD. "Sur la perte des réflexes tendineux dans le diabète sucré," *Progrés Méd.*, 1884, No. 41.
(2) ROSENSTEIN. "Ueber das Verhalten des Kniephänomens beim Diabete mellitus," *Berlin Klin. Wochenschr.*, 1885, No. 8.
(3) LEYDEN. "Die Entzündung der Peripheren Nerven, 1888.
(4) PAVY. "Address on Diabetes," Washington International Congress, 1887, *Medical News* (*Philadelphia*), p. 357, September, 24, 1887.
(5) CHARCOT. "Sur un cas de Paraplégie Diabétique," *Arch. de Neurologie*, 1890, vol. xix., p. 305.
(6) BUZZARD. *British Medical Journal*, June 21, 1890.
(7) ALTHAUS. *Lancet*, 1890, vol. i., p. 455.

(8) AUCHÉ. "Des altérations des nerfs périphériques chez diabétiques," *Archives de Med. experimentale et d'Anatomie Pathologique*," No. 5, 1890.

(9) EICHHORST. "Neuritis diabetica und ihre Beziehungen zum fehlenden Patellar-sehnen-reflex," *Virchow's Archiv*, Bd. cxvii., p. i., 1892.

(10) PRYCE. *Lancet*, July 2, 1887.

Note on cases in which the nerves have been examined microscopically :—

(1) EICHHORST. (*a*) Woman, 45. Death due to coma. No paralysis, knee-jerks abolished, no loss of sensation, no sign of ataxy ; duration two years. (*b*) Woman, 23. Death due to coma. No history ; knee-jerks absent ; muscular weakness of the limbs.

(2) AUCHÉ. (*a*) Paresis of legs, muscular cramps, gangrene of right foot. (*b*) Itching, tingling of feet, legs, and hands ; knee-jerks absent. (*c*) Cramps of calves, diminished sensation on dorsal surface of forearms, subunguinal hæmorrhage and shedding of the nails.

Since receiving the proof of this paper I note that Leyden *(Deut. Medizin. Zeitung*, June 5th, 1893) refers to a case of diabetes with renal disease, in which not only lesions in the peripheral nerves but also in the spinal cord were present. The disease in the cord was not limited to any system. *British Medical Journal*, June 24th, 1893. Epitome, Current Literature.

Clinical Cases.

A CASE OF INTRA-CRANIAL HYDATID TUMOURS WITH HEMIPLEGIA, AND WASTING OF THE PARALYSED LIMBS.

BY J. MICHELL CLARKE, M.A., M.D., M.R.C.P.
Physician to the Bristol General Hospital.

THE patient was a man aged 29, who complained of intense pains in the left temple, of failure of sight in the left eye, and of loss of power in the left arm and leg. He had formerly been of intemperate habits, but during the last three years had been very abstemious. With the exception of syphilis six years previously, he had had no previous illness.

For 18 months he had suffered from occasional epileptiform fits, in which consciousness was lost and the tongue bitten. The weakness of the left side had been coming on for 12 months. Twice during the past six months he had had attacks of pain in the left eye with double vision.

Three weeks before admission he was seized with violent pains in the left temple, radiating over the left side of the head to occipital region; these pains have never left him since then.

He was admitted on January 21, 1893. He was very restless, groaning continually on account of the great pain in his head. He seemed dull and drowsy, but could not sleep, and took no notice of what was going on around him; it was difficult to get a connected account of his illness from him, because of the mental hebetude. He could walk, but only feebly, and dragged the left leg.

On examination the wrinkles of the left side of the forehead were somewhat smoothed out, he could screw up the right eye more tightly than the left, and on smiling the mouth was drawn to the right. Acting separately both angles of the mouth could be well moved. Localisation of sensations, tactile and pain

sensations, were impaired over the left side of the face; thermal sense normal. He protruded his tongue straight. There was incomplete left ptosis; the left pupil was larger than the right, and did not re-act to light. There was optic neuritis of moderate intensity in each eye. There was decided loss of power in the left arm and leg; with the dynamometer the right grasp being 120, the left nil. The power of localising sensations was distinctly impaired over the left side, otherwise sensation over the trunk and limbs was normal. The thoracic and abdominal organs were healthy. There was incontinence of urine, which contained no albumen. The left knee jerk was exaggerated, but there was no ankle-clonus.

On *Jan.* 28, left ptosis more marked, the left external rectus was now paralysed, the optic neuritis had increased, but he had regained some power in the left arm and leg, the grasp on this side being 29.

On *Feb.* 14 the left eye was completely paralysed and immovable, there was cycloplegia and nearly complete loss of vision. Right eye: movement good, pupils acted well; V.=J. 10. He had some convulsive twitchings of the left hand. The intense pain continued and prevented sleep.

On *Feb.* 28, paralysis of the *right* internal rectus and loss of vision over the nasal half of the *right* visual field was noticed. With the left eye he could not detect a ray of light thrown into the eye from the ophthalmoscopic mirror. Muscular wasting in the left arm and leg had been noticeable for some time. Measurements gave R. thigh 17 inches in circumference, L. 16 inches; R. calf $11\frac{1}{2}$ inches, L. calf 11 inches; R. forearm $9\frac{1}{2}$, L. forearm $7\frac{1}{2}$ inches. The electrical reactions, taken on two occasions at a week's interval, showed marked reduction of faradic irritability of muscles of left arm and leg. With the constant current there was increase of irritability. Over the peroneal nerves a contraction was obtained with a current of $8\frac{1}{2}$ milliampères on the right, and 7 milliampères on the left side. There was a similar difference between the two sides over the ulnar nerves. The muscles of the left forearm and leg reacted more readily than those on the right side, and further in the former $ACC > KCC$, $KCC = AOC$. There was thus qualitative reaction of degeneration.

From *March* 4 to 28 a remarkable degree of improvement in the paralytic symptoms occurred. The patient could open the left eye, move the left arm freely, and even feed himself. The mental condition, however, grew steadily worse. He had a convulsion about this time in which the muscular twitchings

began in the left hand, spread next to the left side of the face, and then became general.

On *April* 1 he became very much worse, lay in a stuporous condition, and could not masticate his food. There was now rigid spasm of the left arm and leg. On the 3rd loss of sensation over the left eyeball was noticed, and there was very marked unilateral sweating on the right side of the face, head, and trunk. The head and eyes were turned to the right, and the right pulse was distinctly larger than the left. He died in coma on the 5th; the temperature, which had never exceeded 99° during the progress of the case, reaching 105° before death. The treatment consisted of the administration of iodide of potash in large doses, with mercurial inunctions, and hypodermic injections of morphia for the relief of pain.

At the autopsy a large hydatid cyst, containing several daughter cysts, was found in the posterior part of the left cerebral hemisphere. The cavity measured three inches in the antero-posterior, and two inches in the vertical and transverse diameters respectively. Its walls were smooth and rounded, and showed no signs of inflammatory changes. It thus occupied and replaced the greater part of the white matter of the parietal and occipital lobes. It came nearest to the surface above, under the anterior part of the superior parietal, upper and upper-mesial portions of the ascending parietal convolutions. In this situation the pia mater was firmly adherent to the cortex, tearing it on removal; the grey matter of the cortex was reduced to a layer about $\frac{1}{12}$ inch in thickness. The cortex had undergone most extensive atrophy under that part of the ascending parietal convolution which is concerned in the movements of the arm, and here the ascending frontal convolution was also encroached upon for a short distance. The boundaries of the cyst were sharply defined and surrounded, in all other parts than those first mentioned, by healthy white matter; a broad layer of healthy white substance intervening between it and the cortex.

Another cyst, the size of a large hazel nut, was found at the back of the left orbit, lying between the anterior clinoid process and the sella turcica, just posterior to the sphenoidal fissure and optic foramen, and had compressed and destroyed the left optic nerve and the motor nerves to the left eyeball. All other parts of the brain were healthy. Other cysts were present in the spleen and kidneys.

Unfortunately the extreme upper part of the cervical region of the spinal cord was not reserved for examination, but a large

number of sections of other regions was made, stained in carmine, aniline blue black, and by the Weigert and Pal-Weigert methods, and carefully examined microscopically.

A well-marked degeneration of the left crossed pyramidal tract extended throughout the whole length of the cord. The nerve fibres had largely been destroyed, the sclerosis was of moderate intensity. Sections stained in carmine showed numerous spider cells.

On carefully comparing the two sides in a number of sections the motor cells of the anterior cornu were less in number on the left than on the right side, some had undergone atrophy, others appeared granular and swollen, with loss of their processes. On the left side also more cells were pigmented, and there was a larger amount of pigment in cells thus affected, the nucleus being often obscured. The vessels in the left cornu were more injected, and the peri-vascular sheaths were distended. In some sections there appeared to be a comparative diminution in the fine myelinated fibrils of the grey matter in the left anterior cornu. These changes were more evident in the lower cervical and upper dorsal regions than in the lumbar and sacral parts of the cord. In some sections these alterations in the left cornu were slight, and in most individual sections could be easily overlooked, it was only by comparing the two cornua in a very large number of sections taken from different levels that it became apparent that there was an evident though partial change in the motor cells of the left cornu throughout the cord. When compared with the right side sections of some of the left anterior roots showed disappearance of some nerve fibres. I regret that the examination was not rendered complete by the preservation of some of the smaller peripheral nerves.

Remarks.—Hydated tumours of the brain are rare in this country. Dr. Thomas, of Adelaide, states (*Hydatid Disease*, Adelaide, 1884, p. 124) that 5 per cent. out of a total of 1,987 cases of hydatid cysts were connected with the central nervous system. They give rise to no characteristic symptoms.

In the present case the diagnosis was clear from the localizing signs, first, of a tumour at the back of the left orbit, secondly, of another, and almost certainly larger growth pressing on the motor centres, chiefly on the arm and hand centres, of the right cerebral hemisphere. The growths were supposed to be syphilitic in nature, and from

the usual tendency of syphilitic growths to invade the cortex from without (dura mater), it was thought probable that the tumour arose in this situation. The syphilitic nature of the growths was rendered probable from the fact of the history of previous syphilitic infection, and from the rapid improvement which at first took place under treatment. Further, as multiple intra-cranial growths are practically met with most often as a result of syphilis or tubercle, the absence of any family history of tuberculosis was taken into consideration. An operation was not advisable in the presence of more than one tumour.

A point of interest is the occurrence of wasting and the qualitative reaction of degeneration in the muscles of the hemiplegic side. As is well known in the immense majority of cases of hemiplegia, no such changes occur. When they do occur they are generally considered to be due to some specially irritative influence propagating itself along the descending pyramidal tracts to the motor cells of the anterior cornu. This irritative influence is exemplified in this case by the destructive changes which the cyst had excited in the motor cortex, especially in the part related to the arm and leg centres. The appearances described above show that the muscular changes were due to alterations in the motor cells. The morbid alterations, though slight, are in the whole series of sections quite unmistakeable. Changes in respect of pigmentation have been mentioned above in addition to the more striking signs of atrophy of cells. It is often and justly insisted upon that pigmentation of nerve cells is normal, varying greatly in degree in cells of different parts of the central nervous system. But when the cells of one side of the cord in large numbers contain much more pigment, often to the extent of almost occupying the entire protoplasm of the cell, than those of the opposite side, I think we are justified in regarding it as an abnormal change. As to what part vaso-motor changes may have played in the condition of the cells observed, it is not possible to say. Another point of interest is the total absence of rigidity in the affected limbs until three days before death; one month before death the knee-jerks could not be obtained; they

were only, however, tested in the lying posture. Thus, in the muscular wasting, in the altered electrical reactions, and in the flaccidity of the limbs the hemiplegia in this case differed from that of the ordinary type. These differences are accounted for by the changes in the motor cells of the anterior cornua. Whether we may attribute the morbid condition of these motor cells to the destructive changes taking place in the motor convolutions is more doubtful, but it is interesting to note as pointing to a connection between the two conditions, that the motor convolutions were most affected over the arm and trunk centres and the left anterior cornu in the lower cervical and upper dorsal regions. The connection if a causal one cannot be direct, but is probably an interference of a reflex nature with nutritive processes in the spinal cells.

The distinct loss of the power of localization of sensory impressions in the left arm and leg apart from any other affection of sensation is in accordance with recent experiments on the cortex cerebri.

DIPHTHERIAL PARALYSIS—ALLOCHEIRIA.

BY WILLIAM GAY, M.D., M.R.C.P.

THE following case is chiefly of interest on account of the occurrence of a rare nervous symptom, which, as far as I am aware, has not hitherto been observed in the course of diphtherial paralysis. It consists of a perversion of common sensory impressions to such an extent, that they are localised to an exactly corresponding area on the opposite side of the body. This symptom was first prominently noticed by Obersteiner, who suggested that the condition should be called sensory allocheiria or shortly allocheiria (ἄλλος χέιρ). Such a term is rather misleading, as it might at first sight be imagined to refer to the hands alone, whereas the symptom is most often discovered in the legs.

I have to thank Dr. Christie Wilson, of Doncaster, for handing the case over to me, and allowing me to publish it. M. A., aged 13 years. Father and mother alive, and always in good health. One brother and one sister living and in sound health. No hereditary neurosis. No personal neurotic history at all. The child had just left school to work in a mill, and had always been very strong and healthy. Eleven weeks before I saw her, Dr. Wilson had attended her for a very severe attack of pharyngeal diphtheria, and her condition was regarded as hopeless. She, however, gradually recovered from her original illness, but it was noticed four or five weeks from the commencement that fluids regurgitated through her nose, and that her once shrill voice was reduced to a sort of falsetto whisper. Soon her near vision became deranged and diplopia supervened. The pupils were noticed by Dr. Wilson, widely dilated. There was at this time no actual paralysis, but a generally distributed weakness, so that she was unequal to any exertion and stumbled about in walking. Present condition. Patient is a well developed and healthy looking girl, and makes no complaint except of lassitude. On walking towards me, however, she had to rest her hands on various objects to support herself. Romberg's symptom extremely well marked. She would actually

have fallen, standing with her feet together and eyes shut, if I had not caught her. There was considerable ataxia of all four extremities. No knee, wrist, triceps or jaw jerk. No superficial reflexes. All muscular efforts weak and ill-sustained. Special senses: Eyes—accommodation still defective; pupils dilated, but respond to light and accommodation; weakness of internal recti; otherwise normal. Hearing normal. Smell gone, this was noticed soon after the defect in voice. Taste not tested; but the fact that she could distinguish between some kinds of food, shows that it was probably normal. Common sensation—slight touches not felt at all, but firmer ones referred to exactly corresponding points on the opposite side of the body. This was tested again and again, and created such astonishment in the patient that she would not credit my observation, until corroborated by her parents. She was then astounded to find, that upon taking my hand from her eyes, she had localised a touch upon her ear, face, tongue, arms, and toes, to the opposite side of the body. On only one or two occasions did she hesitate for a moment in so localising her impressions. There was no delay in the transmission of sensory impulses, nor any polyæsthesia. No paræsthesia. Discrimination of heat from cold correct, although there was some slight hesitation in detecting finer shades of difference. Painful sensation not tested. The muscular sense of all four extremities entirely gone. Her limbs were put into all sorts of grotesque attitudes, but the only thing recognised was the warmth of my hand. Movements of head and trunk correctly localised. Voice at present would be best described by the term "cracked," and had by no means regained its old power. Unfortunately, she could not be persuaded to allow a laryngoscopic examination. The soft palate was only slightly paretic, for the b of rub, rub, rub was pronounced like rub, rub, rubm. The condition of affairs was evidently rapidly improving, and in about three weeks from the time I first saw her, she had, but for a feeling of weakness and the loss of knee-jerks, lost all her symptoms.

I was extremely unfortunate in not seeing the case at an earlier period, when all the symptoms were more pronounced. That the essential nature of the affection was diphtherial paralysis can hardly be doubted. The only question that arose was, whether hysteria, which sometimes supervenes, or gets tacked on the symptoms of diphtherial paralysis, was in any way responsible for certain phases of the case, *e.g.*, the aphonia, anosmia and disorders of common sensation. I think not. There was nothing in the hereditary or personal history of the child to favour such a

suggestion, and although some of the symptoms are distinctly unusual in the course of diphtherial palsy, they are with the exception of the transferred sensation, not unknown. I regard the case, therefore, as an instance of diphtherial paralysis, pure and simple.

Laryngeal paralyses are uncommon, but by no means unheard of in diphtherial paralysis. What, however, makes me regret the absence of a laryngoscopic examination the more is that the patient's voice was identically similar to that of a man, a tabetic, who was the subject of ataxia laryngis, and who supplied me with the text for a paper[1] upon that affection, read before the Neurological Society. It will be noticed that there was ataxia of all the extremities, and a certain parallelism between diphtherial paralysis and tabes dorsalis will presently be pointed out. Anosmia is another of the infrequent symptoms met with in this case. It might presumably have been the result of nasal diphtheria, of which, however, there was no evidence. It must, therefore, have been a purely sensory symptom, and equally uncommon in both of the above-mentioned affections.

The most interesting symptom present was, of course, the allocheiria, of which I have been unable to find any example in English literature, although, doubtless, some exist. Obersteiner relates four cases of his own, and five from other German sources, but in no instance was the symptom so widespread or definite as the above. All of them but one, which was hysterical, occurred in the course of tabes dorsalis, which has a close relationship with diphtherial paralysis. Notice, *e.g.*, the loss of knee-jerk at one end of the nervous system, co-existing with ocular symptoms at the other end, the ataxia, loss of muscular sense, &c. The occurrence of allocheiria in the course of diphtherial palsy need therefore excite no surprise. The condition is generally associated with considerable sensory disturbances and loss of muscular sense. The coincidence of these symptoms is best explained by the view entertained by Gowers,[2] that the fibres conveying muscular sensibility correspond with the non-decussating fibres of the postero-median column, with which are mixed decussating fibres from the posterior commissure and neck of the posterior cornu, conveying tactile sensibility. The correct appreciation of temperature is probably explained by the theory that the nerve fibres conveying these impressions are situated in the lateral columns near the antero-lateral ascending tract, which probably conveys painful sensations. According to this view, the

[1] BRAIN, vol. x., p. 494.
[2] "Diseases of the Nervous System," vol. i., p. 133.

discrimination of heat from cold and the appreciation of painful impressions are conducted by non-decussating fibres.

It is extremely difficult to offer any suggestion concerning the pathological state, which gives rise to the condition, allocheiria. Several recent observations go to show that a very intimate connection exists between corresponding sensory areas of the two sides of the body, both physiologically and pathologically. Dumontpellier, for example, by ætherising one arm in a particular position, was able to open painlessly an abscess situated in a corresponding area of the opposite side. Recognising this close sensory relationship between the two sides, may one venture to throw out as a possible hypothesis that, under certain circumstances, when the decussating fibres, together with the non-decussating fibres conveying muscular sense impressions, are, for some reason, put out of court, the remainder of the non-decussating fibres take on the additional duty of transferring common sensory impressions, with which, under normal circumstances, they have nothing to do. If this were so, such impressions would reach the brain on the same side, and would therefore be referred subjectively to the opposite side. For purposes of illustration, one might compare the arrangement with the connection of a telephonic apparatus with telegraphic wires, and the passage along them of vocal messages, for which originally they were not intended, and which, indeed, would never have been sent but for the breaking down of the wires by which they were normally transmitted. This may seem a somewhat extravagant theory, and it may be urged that no connection has been shown to exist between the decussating and non-decussating fibres. On the other hand, it seems exceedingly probable that in the intricate anastomosis of fibres in the cord, there may be some such connection, and the fact that sensory impulses can be conducted, although all motion is lost, when the cord is very considerably disorganised, suggests that when certain fibres are destroyed, their functions can be superadded to those still intact. A similar process must take place in aphasia, for it can hardly be imagined that the third right frontal convolution is a mass of non-functionating material, and yet it may undertake the lapsed duties of the corresponding convolution of the opposite side, and form endless connections with neighbouring centres, *e.g.*, the visual, auditory, arm, &c., in order to carry on the function of speech. It will be noticed in the cases quoted by Obersteiner that, in spite of the grave sensory disturbances, discrimination of heat from cold is still possible, and this function is probably conducted along certain of the non-

decussating fibres of the antero-lateral tract, which must therefore remain uninjured. It will also be noted that, although a heavy touch is referred to the other side, a slight one remains unperceived, and is therefore non-conducted. This rather tends to suggest that there is a certain amount of resistance offered by the new path before it is completely opened out, and that therefore a considerable peripheral sensory excitation is necessary before it is able to overcome this resistance and be conducted to the brain.[1] Gowers mentions a patient, the subject of tabes, in whom a touch was referred to corresponding points on each side of the body at the same time. In this case I should imagine that the decussating fibres were in great part destroyed, but not wholly, and that common sensory impressions found their way up both these and the non-decussating fibres, on account of the relative insufficiency of the former. Such impressions would therefore be referred to both sides of the body.

To recapitulate. All the cases of allocheiria I have been able to find recorded, with the exception of that occurring in the course of hysteria, which sufficiently accounts for anything *bizarre* were accompanied with considerable ataxia and profound disturbance of common sensations. These symptoms are probably due to an affection of the postero-median columns, with the posterior root zones, and include the sensory decussating fibres. The sense of discrimination of heat from cold is probably conducted by non-decussating fibres in the antero-lateral touch. These are as yet unaffected, as in no case was the sense reported lost. The common sensory stimuli, finding their usual channel closed against them, are conducted upwards by the non-decussating fibres, and are consequently referred to the opposite side of the body. This will probably be regarded as very fanciful, and indeed, the theory is only suggested *faute de mieux*.

[1] Diseases of the Nervous System, vol. i., p. 295.

THROMBOSIS OF INFERIOR CEREBELLAR ARTERY.

BY W. F. MENZIES, M.D. ED., M.R.C.P.
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CASES of thrombosis of the cerebellar arteries, causing acute softening, are sufficiently infrequent to merit individual mention.

Ann J., 27, married, was admitted on 17th May, 1890, suffering from early general paralysis.

State on admission.—Physical—Right pupil 5 mm., left 3.5, former inactive to light, latter hypersensitive, both accommodating to distance. The margins are slightly irregular and they are imperfectly circular. Optic discs and retinæ normal. Plantar reflex and patellar jerk dull, the usual facial and lingual tremors present, but not prominent. No ataxia or paralysis, no eruptions, cicatrices or nodes. Copious, dark-red, fœtid lochia, and other usual signs of recent confinement; temperature normal.

Mental.—There is some exaltation and loss of attention, but coherence, rationality, and recent and remote memory are normal. Gives this history: Has been married nine years to a disreputable husband. He deserted her some months ago, and she has since lived with a navvy on the Manchester Ship Canal. Has been somewhat intemperate herself. Pregnancies:—(1) miscarriage, (2) and (3) "7-month" still-born children, (4) and (5) "9-month" still-born children; the last confinement occurred ten days ago.

Progress of case.—Had some transient attacks of delirium during convalescence from confinement, but no rise of temperature. From 1st June till 1st November, 1890, her mental state may, in this connection, be considered normal, and she proved a steady and reliable worker.

Final illness: 1890, *Nov. 1st.*—Complains of severe general headache with gastric irritation, furred tongue, anorexia, no vomiting.

Nov. 2nd and 3rd.—No material change; is quite sensible.

Nov. 4th.—Vomited breakfast, took no more food during the day; has much nausea, headache continues severe. Was wakeful last night. Is weak and giddy, and sways on standing; no true ataxia. Reflexes all depressed.

Nov. 5th.—Does not attempt to stand, saying the head-pain is too severe. Localises it to occiput and forehead. Is dull mentally, but can be easily roused. Utters the "hydrocephalic" moan at intervals. At 4.30 p.m. suddenly jumped out of bed and rushed down the ward, crying out "Murder." Sank down in a heap at the door. The excitement passed in five minutes. Towards evening stupor became prominent.

Nov. 6th.—Lies huddled up in bed, uncomplaining and nearly unconscious. Cannot articulate answers to questions, but mental reflexes remain, and she protrudes her tongue on request. Pupils continue as before, 3.5 mm., nearly inactive to light; pulse 106.

Nov. 7th.—At 9 a.m. drooping of left eyelid was observed, at 11 left ala nasi, at mid-day left side of mouth, and tongue was protruded to left. During the afternoon the furrows of left side of face gradually disappeared. Pulse 115 per minute. Coma not yet absolute.

Nov. 8th.—Some diffuse weakness of left arm and leg. Left plantar reflex and patellar jerk are now in excess. There is slight left ankle clonus. Coma is absolute. Pupils and temperature as before.

Nov. 9th.—Increased flaccidity of left side. Left patellar jerk very active and ankle clonus brisk, right jerk exaggerated, no clonus. Left arch of diaphragm motionless, respiration spasmodic, no cyanosis. Temperature began to rise towards evening; at 7.30 was 99.6° F.

Nov. 10th.—General muscular flaccidity, all reflexes absent, respiration irregular, gasping and shallow; cyanosis and sweating present. Temperature at 7 a.m. 99° F., at mid-day 99.8°, at 4 p.m. 101.4°, at 8 p.m. 103.4°. Thence it fell until death at 10.30 p.m.

Post-mortem 13 hours after death.—Moderate rigidity. We may summarise that the brain was firm, little wasted, and showed the usual signs of early general paralysis, that the vessels were widely affected with endarteritis, but showed no atheroma, and that other syphilitic lesions were absent. The outer and posterior aspect of the inferior surface of the left lobe of cerebellum was extensively softened and pulpy, and the condition extended at least 2 cm. into the substance of the organ. A like condition was present in the left inferior cerebellar peduncle, and extended down the cord to the middle of cervical enlargement, gradually

lessening in degree and extent. On the left inferior cerebellar artery, 1 cm. from its origin from the vertebral, was a sclerosed patch 4 mm. × 2 mm. Beyond this the vessel was empty, but a clot spread from it to the vertebral, and thence down the latter vessel as far as the foramen magnum, entirely blocking the lumen. At first it was firm and partly decolorised, but lower down quite recent. The vertebral above the origin of the inferior cerebellar was free and empty. The left arch of diaphragm reached to the upper border of the fourth rib, two spaces higher than usually found. The right was in the ordinary position, reaching to the upper border of fifth.

Remarks.—This case is incomplete, because the cerebellum, medulla and cord, with the vessels, which had been set aside to harden, were accidentally thrown away, before sections were cut. No opinion, therefore, can be expressed as to the limits of the lesion, the centres affected, or the presence of other vascular obstructions. But the clinical aspect remains, and possesses a certain negative interest in supporting the ordinarily accepted theory that lesions of the lateral lobes of cerebellum produce, *per se*, no evident diagnostic symptoms. We may assume, from the patient's own account of her pregnancies and habits of life, and the evidence on admission of her having lately given birth to a dead fœtus (copious putrid discharge without septic absorption), even in the absence of an independent history, that she was syphilitic; and shall probably be correct in referring the arteritis and general paralysis to this cause. But further deduction is unsafe. Thus, the headache was at first supposed to be functional, due to over-eating; and when, three days later, its organic nature seemed likely, the mental torpor was already too pronounced to permit of an exhaustive search for psychical changes of any kind. The pupils and other reflexes were put out of court from the first, owing to the presence of general paralysis. It is probable that the localising signs which appeared on the sixth day of illness denoted the advance of the clotting process from the inferior cerebellar along the vertebral, interfering with the nutrition of the medulla, and that the fatal event was retarded for three days owing to the free anastomoses chiefly through the anterior spinal. It will be noted that the 7th and 12th

nerves were earliest affected, and that evidence regarding the 8th, 9th, 10th, and 11th, is indeterminate. The diagnosis was arachnoid hæmorrhage (the so-called "pachymeningitis hæmorrhagica interna") on the inferior surface of cerebellum, spreading down the side of the medulla, and exerting pressure on the nerve roots, and later, on the columns. Probably the extreme rarity of that lesion in this situation should have prompted a more correct guess.

POLYURIA (DIABETES INSIPIDUS) IN BASAL CEREBRAL DISEASE.

BY HENRY HANDFORD, M.D., M.R.C.P.LOND.
Physician to the General Hospital, Nottingham.

THE following two cases are the only examples of this phenomenon with which I have met in a somewhat extensive hospital experience. And the second, in which the fact rests only upon the statements made by the patient and her friends, is sufficiently indefinite to be cast aside, were it not so amply supported by the details of the first case.

Gowers says, "The urine is seldom altered in composition by organic brain disease, although polyuria rarely, and glycosuria occasionally, have been produced by disease of the pons and medulla oblongata."

The analogy with diabetes from puncture of the floor of the fourth ventricle seems to be very close. It would appear probable that the pressure of a gumma paralysed the vasomotor centre for the kidney in the medulla, the absence of polyuria until the latter part of the second attack, and its continuance when all other symptoms were improving, is difficult to explain. But we must accept as a fact that a tumour may grow all round a nerve without pressing upon it sufficiently to interfere with its function, and that of several nerves to all outward appearance equally involved and pressed upon by a tumour some may be completely paralysed, and others quite uninjured. The direct and indirect pressure upon the brain substance is also open to many sources of variation. Hence it is useless to speculate upon the escape of the renal centre until a particular time, and the absence, or slight degree of, improvement when the other symptoms of pressure upon the nerve trunks and nerve tissues were so greatly diminishing.

The frequency with which iodide of potassium and mercury are given for diseases of all kinds without any such diuresis following, renders it clear that the treatment was not in any way responsible for the phenomenon.

I have detailed the first case very fully in order that it may bear internal evidence of the accuracy of the diagnosis, both as to the presence of a tumour and as to its syphilitic nature.

Case 1.—Julia M., aged 27, lacewinder, admitted into the General Hospital, Nottingham, Oct. 15, 1891, complainng of fits, &c.

Her father died in the hospital of chronic Bright's disease. Her mother died of dropsy: brothers and sisters are healthy. Patient had fits as a child, and five years ago fell and struck her head, remaining unconscious for two or three hours. She completely recovered. Before marriage she was for some years a prostitute; had venereal disease, rashes, and sore throat. She has been married 6½ years, and has two children, the youngest eight months old.

Her present illness commenced about eight months ago, three weeks after her last confinement. She noticed that her left eye was getting "crooked," and she frequently vomited. (The vomiting continued till six weeks ago).

She next found that her left arm and leg were getting weak. She could not hold the baby on her arm.

She suffered from double vision, headache, and a feeling of pain, and "pins and needles" shooting down the left side. This peculiar numb feeling usually only continues about five minutes, but when it lasts a quarter of an hour or longer she loses consciousness. She can tell when these "fits" are coming on.

On admission she was found to be well nourished and fairly intelligent, but her memory was evidently failing.

The grasp of the left hand is markedly weaker than the right: —dynamometer, L. 35, R. 65. The left leg is also evidently weaker than the right. There is no facial paralysis, and the tongue is protruded straight. Sensibility to touch and to pain are normal, except perhaps over the left arm, where they are both somewhat deficient. The muscular sense is impaired on the left side. Sensation to heat and cold normal. Taste impaired, but not lost.

The plantar reflexes are exaggerated, especially on the left side; the patella reflexes also; no ankle clonus. The left wrist, triceps and biceps jerks are exaggerated.

There is complete paralysis of the right external rectus; and marked weakness of the left external and internal recti. There is nystagmus on the left side, and some proptosis. No optic neuritis, but masses of pigment showing remains of old choroiditis. Left eye myopic, right hypermetrophic.

Ten days after admission the note runs:—Since admission she has had 3 to 9 fits daily, never losing consciousness. She has vomited once or twice for the past two days. The headache has been more severe, and is general in distribution. It is better at night and allows her to sleep. She is up in the afternoon and can get about.

Sensory Aura.—She always knows when the fits are coming on by a pain in the left temple, followed by a feeling of "pins and needles" in the left arm, commencing in the fingers and passing up the arm. This is from five to ten minutes before the fit commences. After the "pins and needles" in the arm comes a pain in the left flank just below the tenth rib, passing down the outer side of the thigh and leg to the toes which are then flexed, and immediately the leg becomes rigid and remains extended. She then rubs the left foot with the right. In the meantime the fingers of the left hand are flexed, and the arm becomes extended until she grasps the wrist with the right hand and draws it across the chest. She does not utter any cry. The fit lasts about two minutes. The mouth is drawn a little to the left. The procedure is exactly repeated every time. She becomes drowsy after the fits, and complains of the headache becoming much more severe and general.

Nov. 2.—She has been taking iodide of potassium, gr. x., three times a day, and pil. hydrargyri gr. $3\frac{1}{4}$, twice a day, ever since Oct. 20. She is now gaining power in the left arm and leg, and the fits are not nearly so frequent. The aura has ceased, so that now the fits come on without warning. And instead of being preceded by tingling in the leg, they are *followed* by pain in the left middle finger going up to the elbow. The pain in the head has been much less severe.

Dec. 2.—For the past few weeks she has never had more than one slight fit daily and often none for several days.

Now the fits *commence* in the leg with rigidity of the muscles; the tingling and sensory phenomena have ceased.

Jan. 8.—She has been away to the convalescent hospital for a fortnight, and during that time has only had one fit.

The left leg seems now as strong as the right, and she can walk several miles. There is no stiffness, but the left knee-jerk is greatly increased.

She has had no headache for several weeks. The reactions of the pupils are normal, but the left is slightly larger than the right. There is still weakness of both external recti and nystagmus on abduction. No change in the fundus besides the remains of old choroiditis. She is now an out-patient.

Jan. 21.—Since she left the hospital a perforation of the soft palate has formed close to the uvula, which seems likely to slough off. There is also a syphilitic pharyngitis.

March 7.—Patient was readmitted. She had not attended since Jan. 21.

She has had much headache and vomiting; and is now very feeble and emaciated. She is hardly able to stand, and usually lies in a semi-comatose state. The left knee jerk is exaggerated, the right almost absent. There is no distinct clonus on either side, but some slight rigidity of both legs. Slight impairment of common sensation over the right hip and flank. The condition of the palate and fauces is unaltered since Jan. 21st. She can move the tongue freely: it is dry and coated with a brown fur. Temperature 97.2.

There is complete paralysis of the left external rectus, and nearly complete of the right, Pupils more than half dilated. Left disc swollen: vessels curving over the edge. Right disc slightly hazy. Left globe prominent.

April 4.—Patient has been treated by iodide of potassium, gr. x, three times a day, together with mercurial inunction.

For a few days after re-admission she was delirious, especially at night; and for a week or two she lay in a semi-conscious, drowsy, state; for the first fortnight the urine was scanty.

From *March* 15 the quantity of urine almost suddenly began to increase enormously, ranging from 100 oz. to 130 oz. in addition to what was lost with the motions. The sp. gr. varied between 1,006 and 1,008; and the urine was quite free from albumin and sugar.

From *April* 12 to May 2 patient again went to the convalescent hospital and improved in general condition, but the urine still continued between 100 and 140 ounces daily, besides what was necessarily lost.

On *May* 25 the iodide of potassium was stopped, and ʒi. of liq. hydrargyri perchloridi alone given. The quantity of urine was not materially affected, although it continued to be carefully measured for ten days further. She was then made an out-patient.

A month later, June 22, she was again re-admitted for two days, and the polyuria still continued. The paralysis of the

external recti, the perforation of the soft palate, and the exaggerated knee jerk remained. Otherwise she seemed well. She was advised to continue medicine once a day for another six months; but she did not return, and has been lost sight of.

The second case is not nearly so definite. She was admitted in January, 1890, under Dr. Ransom, to whom I am indebted for permission to use the case.

The patient was a woman, aged 34, who had had two children and three miscarriages, with a very definite history of syphilis.

During the past year she has suffered much from headache. In April, 1889, *i.e.*, nine months ago, she began to suffer from intense thirst. In one afternoon she drank two dozen half-pint bottles of soda water. She did not drink intoxicating liquors. In one day she has passed as much as three gallons and half a pint of urine.

This polyuria ceased two months ago, when the headache became more severe, and drooping of the left eye-lid began. She has had no vomiting.

She is slow in answering questions, and seems to forget words. There is no paralysis of face, arms, or legs; but there is complete ptosis of the left eye-lid, and the left pupil is dilated and does not re-act to light or accommodation. No optic neuritis. Sensation is unaltered. She has had several slight epileptic seizures.

She was treated with pot. iod. gr. x., three times a day, and improved considerably. The polyuria did not return while she was in the hospital. A gumma was diagnosed in the temporo-sphenoidal lobe, involving the third nerve as it emerges from the base of the brain.

A CASE OF FRACTURE—DISLOCATION OF SPINAL COLUMN, WITH LESION OF THE CORD.

BY F. W. TUNNICLIFFE, M.B.LOND.
Late House Surgeon, St. Bartholomew's Hospital.

FRACTURES and fracture dislocations of the spinal column with consequent lesion of the cord being of fairly common occurrence, perhaps some excuse will be expected for publishing this one. The chief interest of the case, I think, lies rather in the completeness and suggestiveness of the symptoms and in the microscopic morbid anatomy than in the trauma, and this is my reason for wishing to publish the case; for permission to do this, as well as for supervision throughout, I am indebted to Mr. Bruce Clarke.

J. M., aged 36 years, a builder, was admitted into Darker Ward, under the care of Mr. Bruce Clarke, on Jan. 27, 1892, suffering from a fracture dislocation of the spinal column.

History of present condition was as follows:—

On the afternoon of admission patient fell 30 feet from the top of a ladder, and when on the ground the ladder fell on his back.

Family history and past history both irrelevant.

Patient is a big heavily-made man, height 6ft. 2in.; upon admission, temperature 98° F., pulse 80; evident signs of dyspnœa.

Upon examination a lump was found in mid-dorsal region, with a hollow immediately above, this projection was apparently formed by the 6th dorsal vertebra, and its prominence was due to the fact that the column above was displaced forwards.

The following marked functional disturbances were present:—

A. Below line of lesion.

(1) *Sensation.*

Complete loss, including loss of sensations of—

(*a*) Touch.

(β) Pain.

(γ) Heat and cold.

(2) *Motion.*

(α) Complete paralysis, respiration entirely diaphragmatic.

(β) Idiomuscular twitchings of great pectoral muscle.

(γ) Retention of urine.

(3) *Trophic.*

Factitious urticaria, tâche cérébrale could be produced anywhere below level of lesion.

(4) *Sweating.*

Sensible perspiration absent below lesion.

(5) *Reflexes.*

(α) Cutaneous reflexes, plantar and cremasteric, *present.*

(β) Deep reflexes, patella, ankle clonus, *absent.*

B. Above line of lesion.

(1) *Sensation.*

(α) A narrow zone of imperfect sensation, tactile, pain, heat and cold feeling being equally diminished.

(β) A zone, about 3 inches wide, of marked hyperæsthesia, the slightest touch causing pain.

Application of a test tube containing water at about 90° F., sensation of scalding.

(2) Motion normal.

(3) Sweating profusely.

(4) Deep and cutaneous reflexes normal.

Vide Diagram.

Urine drawn off with a catheter (no uretheal spasm) acid 1025, no albumen, no sugar.

Temperature upon admission normal. Pulse and respiration also normal.

In addition to the spinal column injury, there existed also a fracture dislocation of the manubrium sterni.

The manubrium, with the two first ribs, being displaced backwards and downwards. No fracture of the ribs was made out.

The treatment consisted in making extension, under chloroform, on the shoulders and head, while the pelvis and the feet

were fixed, with also at the same time manipulation at the seat of fracture: this succeeded in reducing partially, but not entirely, the dislocation; the extension and counter extension were performed with the patient on his side, he was then rolled over

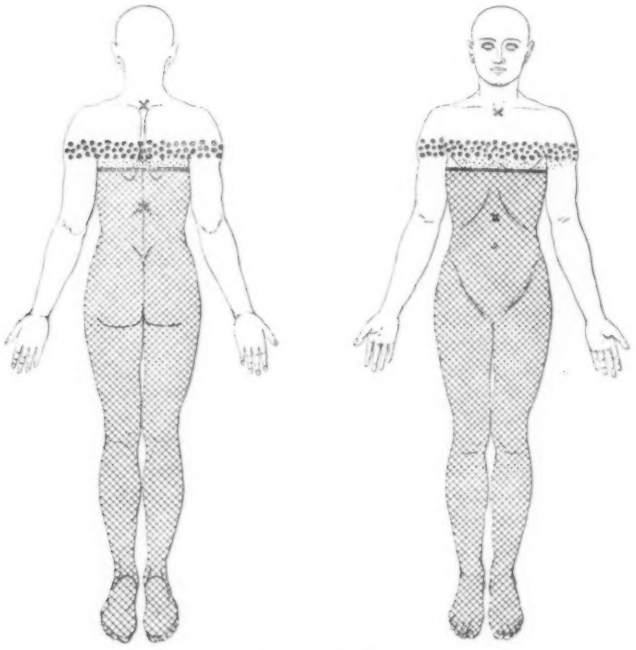


DIAGRAM I.

——— Line of lesion.
× × 7th cervical and 1st lumbar vertebræ.
∴∴∴ Zone of hyperæsthesia.
..... Zone of imperfect sensation.
xxxxxx Region of complete anæsthesia and paralysis.

on his back on a water bed. His head was kept in a horizontal position, and was prevented from moving from side to side by sandbags.

Jan. 28.—Patient passed a very restless night. Morphine gr. $\frac{1}{4}$ administered. Respirations rapid (32 per minute) and laboured.

Temperature 99° in axilla. Patient still unable to pass urine. Catheter passed every 6 hours.

Jan. 29.—Dyspnœa more marked. Resp. 40. Patient is becoming cyanosed. Pulse 120. Temperature 10° F.

Patient continued to get worse rapidly. Death occurred 2 a.m. Temperature at time of death 106.6° in rectum, 102·6° in axillæ, 102.8° in mouth.

The *post-mortem* examination made the day of death, Jan. 30, revealed the following conditions :—

Both pleuræ contained large quantities of blood. Contents of pericardium normal. No rupture of large thoracic vessels made out, hæmorrhage probably arose from rupture of internal mammary veins which was dependent upon the fracture dislocation of the manubrium sterni.

An oblique fracture was found through the manubrium sterni ; direction of fracture was from above, downwards and backwards. The upper piece was driven backwards and downwards, and the two first ribs with it.

Spine.—The laminæ of the 4th, 5th, and 6th dorsal vertebræ were fractured, on opening canal there was some blood outside theca, but not enough to cause pressure on the cord, within the theca there were no clots visible. The cord was entire as regards its continuity ; at the level of the 6th dorsal vertebræ cord was swollen and bluish. The bodies of the 4th, 5th, 6th dorsal vertebræ were fractured.

Microscopical Examination of the Spinal Cord.—The portion of the cord corresponding to the seat of injury was hardened in Müller's fluid, embedded in celloidin, and the sections were stained in logwood and eosin. Portions were also excised from the cervical, and lumbar regions of the cord, and were examined in the same way.

A few sections from the centre of what appeared to be the most bruised part were chosen for microscopical examination, and then a few sections from the upper and lower limit of this until at either extremity healthy cord was reached. The effect of staining with logwood and eosin, for which suggestion I am indebted to Mr. Austen, is to stain all hæmorrhagic patches dull red, while the rest of the cord is colored bright purple. The sections upon microscopic examination shewed that the cord had been crushed. The central canal was no longer recognizable.

The entire grey substance was the seat of scattered patches of hæmorrhage, the hæmorrhage extending along the anterior and posterior nerve roots. Of the white matter, the posterior and postero-lateral columns were most affected.

Upon the use of a higher power the individual eosin stained blood corpuscules composing the dull red patches could be distinctly recognized. Sections above and below this, the most injured level, shewed a gradual diminution in extent of the hæmorrhages, this could be traced section by section until healthy cord was reached.

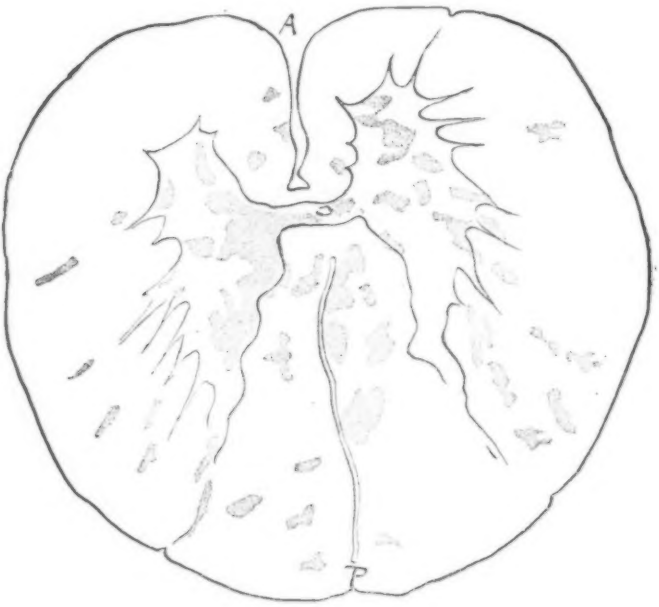


DIAGRAM II.

A anterior, P posterior, longitudinal fissure of the cord.

The result of the microscopical examination of the lumbar cord at the level of the lumbar enlargement was to confirm the macroscopic appearance, viz., to show nothing abnormal.

The adjoining diagram illustrates a section, taken from the centre of the bruised portion of the cord. The shaded areas mark the positions and extents of the hæmorrhages. For this diagram, as for the preceding ones, I have to thank Dr. Adamson.

Remarks.—There is, I venture to think, considerable interest in this case on account of, at any rate, two phenomena. I refer to the absence of sweating below the

lesion and the high temperature in the rectum, as compared with the axillæ and mouth. I will not venture to discuss a phenomenon which is also interesting, viz., the complete abolition of the deep reflexes below the lesion, this I am aware can be attributed to shock. I will merely remark, *en passant*, that the cutaneous reflexes were present, also that the so-called "shock" was accompanied by stimulation of the vaso-constrictors, as evidenced by the dryness and pallor of the skin below the lesion. The idea of nervous shock, although the turn is as indefinite as it can be, is usually associated with that of paralysis, of dilation of the vessels of the skin, in short with sweating; here at any rate this was not the case, there was moreover, no evidence of paralysis of the sphincter ani or vesicæ, in other words there was no evidence of "shock" below the lesion other than the abolition of deep reflexes.

It is also to be noted, in view of recent[1] theories upon this subject, that no myelitis existed elsewhere, more particularly that the lumbar enlargement was healthy.

With regard to the profuse sweating above and the entire absence of sweating below the lesion, I know of nothing in any way similar.

It must also be remarked that the skin was very much redder above than below the line of lesion; in other words, there was dilation of dermal vessels. This profuse sweating, which was much more marked during sleep, cannot be attributed to the temperature, this being only 99° in axilla and mouth. Dr. Brunton[2] has sought to attribute the night sweats in phthisis to the increased amount of carbon dioxide in the blood, which itself is due to the diminished respiratory capacity of the individual. Leaving out of consideration why they occur at night, the fact remains that the respiratory capacity of the patient in question was suddenly considerably diminished, by the occurrence of a double traumatic hæmatothorax. This taken in connection with the fact that the patient's sweating area was lessened would probably explain the diaphoresis above the line of the lesion.

[1] Gowers. Spinal Cord, 2nd edition. Lond., 1892.

[2] *St. Bart.'s Hosp. Reports*, vol. xv., "Pathology of Night Sweating in Phthisis."

The most interesting phenomenon remains, viz., the complete absence of sensible perspiration below the line of the lesion, this being undoubtedly accompanied by constriction of the vessels of the skin, as evidenced by the surface temperature (a surface thermometer was not used) and the paleness of the skin. The question naturally arises, was the absence of sweating entirely due to the diminished blood supply, in other words, was it merely the expression of a vaso-motor change, or must we look to some concomitant change in the origin or centres in the cord of the secretory nerves distributed to the sweat glands. We know perfectly well that skin pallor can occur with diaphoresis on the one hand, as in fright, nicotine poisoning, &c., and also skin redness with complete absence of sweating, as in atropine poisoning, on the other, thus shewing distinctly that the two mechanisms, although generally acting together, can each when occasion requires, act separately. We must, I think, conclude that both causes were here at work to cause absence of sweating.

With regard to the temperature, unfortunately the only comparative observation was taken just before death, this shewing a difference of 3.8° between rectal and mouth temperature at the same time. Whatever this may mean, one cannot help noticing that the high temperature was like the absence of sweating, below the lesion, in other words, that relatively to the lesion in the cord, high temperature was associated with diminished heat loss.

A CASE OF INJURY TO THE MEDIAN NERVE ILLUSTRATING THE POWER OF REPAIR OF A MIXED NERVE TRUNK; AND THE MODE OF ACTION OF PILOCARPIN.

BY THOMAS D. SAVILL, M.D.

In Infirmary practice one often has the opportunity of observing a patient over a long period of time, and the following case, which has been under my notice for nearly six years, may be interesting.

It illustrates the power of repair of a mixed nerve trunk, where the injury is of a very definite and known extent.

Daniel Flynn, who was then 20 years of age, a bargeman, was admitted to the Paddington Infirmary on September 20th, 1887, for tetanus. The case was one of extreme severity, and as there was a clear history, 14 days before admission, of injury to the palmar twigs of the median nerve by falling on a dust heap, it was thought that division of the nerve might possibly give him an additional chance of life.

Accordingly upon *September 28th*, 1887, *half-an-inch of the median nerve trunk in the upper arm was removed, the two ends being loosely connected by cat-gut, leaving* $\frac{3}{8}$ *of an inch interval.* The wound healed by first intention; and this operation was, of course, followed by paralysis of the muscles, and anæsthesia of the skin supplied by the median nerve. These symptoms, it was hoped, would pass off after the nerve had re-united. But it was not so; the weakness of the muscles increased, they became atrophied, and presented marked reaction of degeneration.

At the end of three months (*Dec.*, 1887) blebs formed spontaneously on the knuckles of index and second fingers of the right hand. These blebs soon became open sores (and did not heal for any great length of time during the next 13 months, fig. 3). About this time, also, it was noticed that the skin over the outer half of the hand was rougher and redder than that over the inner,

and that this half of the hand perspired a good deal more than the other, pointing to a vaso-motor lesion. He was treated with

FIG. 1.

Shows area of complete anæsthesia, December 22nd, 1888.

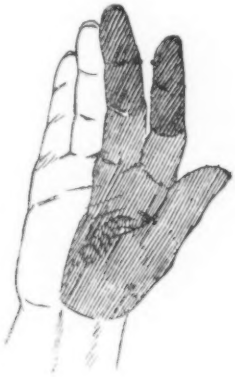


FIG. 2.

Shows area of complete and partial anæsthesia, May 7th, 1888.

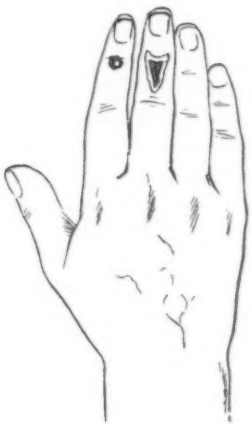


FIG. 3.

Sores on knuckles, May 7th, 1888.

galvanism, massage and faradism without result. The accompanying sketches made for me by Dr. G. W. Davis, at the bedside,

show the state of the anæsthesia at that time. It will be noticed that in May, 1888, the area of complete anæsthesia extended only just above the first phalangeal joint.

In *June*, 1888, eight months after the original section the continuity of the median nerve had been restored to some extent, for slight power of movement had returned in the fingers, and acute pain was felt on pressing over the nerve trunk in the fore arm and in the upper arm below the injury. But the nerve was obviously the seat of some irritation, and I therefore decided to cut down and explore the nerve trunk at the seat of the original operation, where a swelling in the nerve could be felt.

FIG. 4.

Shows maximum closure of fingers and thumb, September, 1888, one year after the first, and three months after the second operation (resection). Also shows atrophy of muscles of thenar, wrinkled skin over outer half, and position of bleb on knuckle. The muscles of the forearm were also much atrophied.

On *June 8th*, 1888, one inch and a half of the median nerve trunk including a fusiform fibro-neuroma was removed, and the healthy cut ends brought together by two cat-gut sutures as closely as it was possible; that is to say, within half-an-inch of each other. The wound healed by first intention. The limb was put up in plaster with the forearm semi-flexed, and kept so for ten weeks.

For several months after the operation the patient suffered a great deal of pain down the median nerve, which was so much increased by extending the arm that it had to be kept flexed. Three months after the resection (*September*, 1888) motor power began to return. The accompanying sketch (fig. 4), from a photo-

graph made at that time shows the maximum power of closure of the fingers and thumb, and other particulars, three months after the second operation. There was considerable atrophy at this time, not only of the flexors of the forearm and the thenar muscles, but also of the bones, for there was a shortening and thinning of the fingers; the photo also shows the wrinkled state of skin on the outer side of the palm. Fresh sores formed, this time on the tips of first and second fingers. Sensation had not returned; it was now the same as shown in fig. 2.

With a view of ascertaining whether pilocarpin really excites sweat through the sweat nerves, the following experiment was performed on *Nov. 9th*, 1888.

"11.10 a.m.—Condition of hand as usual—inner side sweating, outer side slight blush. Pilocarpinæ nitratis gr. $\frac{1}{10}$ injected into *left* arm. In five minutes the outer side became more red than inner side, but no alteration of perspiration. In ten minutes no further effect, so $\frac{1}{10}$ gr. more injected.

"In fifteen minutes outer half of palm (*i.e.*, part supplied by median nerve) very red, much redder than inner side.

"Inner side perspiring profusely, outer side not at all.

"Profuse perspiration all over body."

In *February*, 1889, eight months after the resection he left the Infirmary. At this time power had, to some extent, returned in the muscles (dynamometer—right 39, left 78), and he could close the fist completely, though the movements were stiff. There was pain experienced at the seat of operation, stiffness on extending the forearm, and tenderness down the median nerve. The sores on the fingers had quite healed now. The atrophy, condition of skin, and anæsthesia all remained the same.

June, 1889.—He went to a convalescent home for some months, and on his return to the infirmary in *June*, 1889 (one year after the resection), power of motion had still further improved (dynamometer—right 51, left 85). About this time sensation began to improve; but atrophy still remained the same.

In the *Autumn of* 1889 (two years after the original operation and one-and-a-quarter after the re-section), the muscular atrophy began to improve, and in March, 1890, there was only $\frac{1}{2}$ inch difference in the measurements of the two forearms.

At the present time, *June*, 1893 (five years after the resection), there is still some deficiency of power (dynamometer—right 52, left 86), and also slight stiffness; but he has been able to follow the occupation of omnibus conductor during the last three years with perfect success, excepting that he has to use his left hand to

sort the money in his bag on account of stiffness of the first and second fingers of his right hand. There is no anæsthesia or analgesia now; he distinguishes heat and cold, and his muscular sense is good. The skin of the outer half of right hand is now paler than the corresponding part of left, there is still some tenderness over the course of the median nerve in the forearm, and still scars from the old blebs on knuckles and tips of fingers.

As regards nutrition of muscles, the two forearms now measure precisely the same, and the two thenar eminences are the same, showing that the muscles have fully regained their volume. But it is not so as regards the bones; there is $\frac{1}{4}$ inch difference in the circumference of the two wrist joints; and the right thumb, first and second fingers are measurably shorter and thinner than those of the other hand.

The following measurements are in centimetres :—

Right.		Left.	
Wrist	15.8	Wrist	16.5
Round distal joint of thumb	6.1	Round distal joint of thumb	6.5
„ „ „ index finger	4.6	„ „ „ index finger	5.2
„ „ „ second „	4.8	„ „ „ second „	5.2
Total length of thumb	6.5	Total length of thumb	7.0
„ „ „ index finger	9.5	„ „ „ index finger	9.5
„ „ „ second „	10.8	„ „ „ second „	10.8
Round hand opposite cleft of thumb	20.6	Round hand opposite cleft of thumb	22.4

Remarks.—The injury at the time of resection (in June, 1888) was very considerable, and was of well-known extent; $1\frac{1}{2}$ inch was removed. The nerve trunk on the peripheral side was already in a state of degeneration (as shown by the microscope). The ends could not by employment of any reasonable force be brought together closer than $\frac{1}{2}$ an inch.

The chief points of interest are :—

(1) The extent of recovery in the muscles. They have now (June, 1893) quite resumed their former volume, and very nearly their old strength. The patient only complains of a little stiffness in the fingers.

(2) The permanent atrophy of the bones.

(3) The complete restoration of sensation.

(4) The pilocarpin experiment, producing extreme redness over the palmar branches of the median; but no perspiration, such as elsewhere occurred, over this part of

the hand. It shows that this drug acts through nerves conveyed in the median trunk.

His remarkable recovery from tetanus, under treatment by large doses of chloral hydrate, was the subject of comment elsewhere.[1]

The recovery of the nerve may, to a large extent, I believe, be attributed to the limb having been kept at perfect rest.

I was inclined at one time to take a very gloomy view of the prognosis, and contemplated the transplantation of nerve from an animal, as in Gersung's case.[2]

But on reading in Hilton's "Rest and Pain" of three cases of injury to nerve which recovered after several years' rest, I prescribed this treatment for my patient, and the result has been most satisfactory.

[1] *Lancet*, November 24, 1888. [2] *Brit. Med. Journal*, May 19, 1888.

Abstracts of British and Foreign Journals.

Model of the upper part of the Spinal Cord and the Oblongata.[1] By Dr. Ludwig Edinger, Frankfort-on-the-Maine.

The knowledge of the inner structure of the central nervous system is generally acquired by examining and combining transverse sections. As the delineations of these impress themselves especially on the minds of students, I have found it advisable, in my new model, to represent the fibres as issuing from the reproduction of a transverse cut.

A revolving pillar rises from a heavy pedestal; to the top of this is attached a horizontal bar—75 cm. long—representing the central canal. To one end of the bar is fastened a perforated metal plate on which the rhomboidal area of the medulla is painted, whereas the other end of the bar terminates in a similar reproduction of the cervical part of the spinal cord. The various nuclei and fibres are distinguished from one another by different colours. The proportions as well as the colouring have been so selected that minute details can be seen at a considerable distance.

The most important fibres and cells have been reproduced by means of wires and wooden models. Between the cross sections a number of variously painted wires are extended which represent connections of identical areas or continuation of the entering root fibres.

The cells of the spinal ganglia and of the motor and sensory nuclei are shown in their various forms. Larger aggregations of cells, of which it seems difficult to give a proper imitation in a model, as for example the forms of the nuclei of the posterior columns and the ascending gelatinous column of the trigeminus and glossopharyngeus are simply given in carved wood. In order

[1] Author's translation from his paper in *Anatomischer Anzeiger*, 1893, No. 5.

not to make the matter too complicated the nuclei of the hypoglossal and vagus are given in the same form.

As the model is designed to give a first insight into the subject, I have endeavoured to make it as simple as possible, and several tracts consisting only of a few fibres have been omitted. The name of each part is signified by a printed label. (In the wood cut, which illustrates this description, numbers are substituted.)

As few colours as possible have intentionally been used, and each tract retains its colour throughout. All motor root tracts are painted light red, the primary sensory tracts light blue. The secondary motor tracts are dark red, the central sensory tracts dark blue.

All the wires representing tracts which extend either caudad or frontad beyond the sections project at either end.

The model was completed in the spring of 1892, and shows matters as they were treated in the third edition of my "Vorlesungen ueber den Bau der nervösen Centralorgane," Leipzig, C. W. Vogel.

In studying the model it is the best plan to begin with an anterior root (light red wire, No. 32 in the cut). Motor roots enter into the transverse sections as well as into the intermediate part of the model, but only the cross section gives a complete view of the different ways they take. Every root fibre terminates in a multipolar ganglion cell. Round this cell twines a branch of fine red wires, the extremity of the secondary motor tract (red wires) which springs as a collateral either from the lateral (No. 26) or anterior (27) pyramidal tract. It is easy, in this manner, to follow these pyramidal fibres as far as the base of the medulla where the lateral tract decussates and both together form the ventral pyramidal area (No. 19) of the oblongata.

Of the sensory nerve (light blue, No. 30) a short peripheric portion is given, the greater part of whose fibres terminate in cells of the spinal ganglion (31). From these cells the roots enter the model and divide in the well known manner into different branches. Each part, however, is here represented by a single fibre only. The division of entering roots into ascending and descending branches is shown. One part of the posterior roots enters the posterior tracts (20, 21) in which it ascends and can be traced as far as the posterior nucleus funiculatus of the medulla. Another reaches the grey matter through the posterior grey column where its fibres either spread themselves around the cells or proceed to the Clarke's column. One fibre can be traced up to a cell of the anterior grey column. The direct tracts leading

to the limiting layer of the grey substance as well as the fibres extending to the crossed posterior tract have been omitted; likewise the posterior commissure, because our knowledge of the structure of the latter is too imperfect.

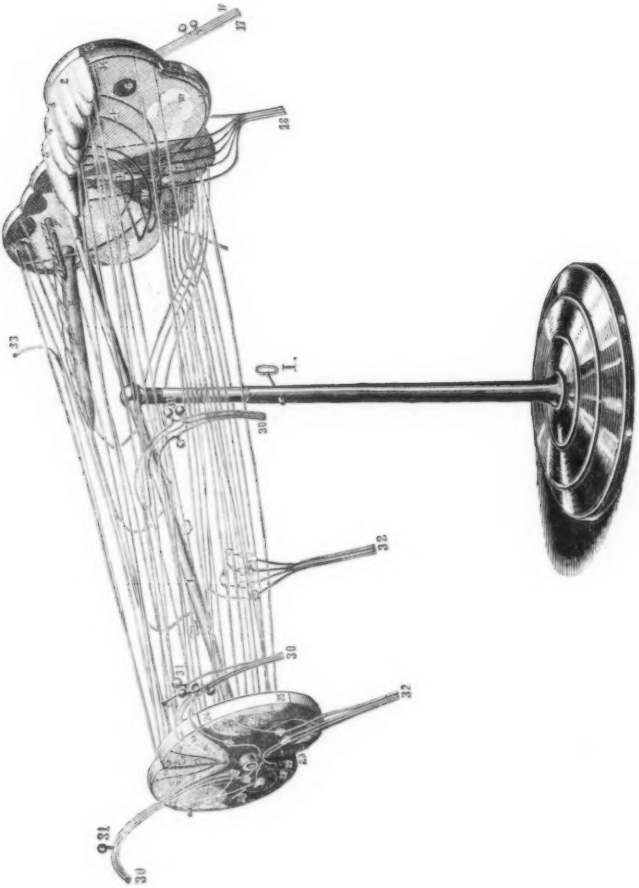
From the fusiform cells (dark blue) of the posterior column springs the secondary sensory tract (dark blue). It decussates through the anterior commissure into the antero-lateral tract, and may be followed up to the fillet (11). The (yellow) fibres of the lateral cerebellar tract spring from the cells of Clarke's column. 25 shows the position of the fasciculus antero-lateralis at the central margin of the above-mentioned lateral cerebellar tract. Lastly, in the spinal cord a single commissural cell is shown which sends its dividing fibre to the antero-lateral tract of the opposite side.

The secondary tracts which spring from the nucleus of the funiculus cuneatus and funiculus gracilis in the medulla are the following: On the mesial side the internal arciform fibres (15) crossing to the fillet, and laterally the external arciform fibres anterior and posterior (13, 14) to the restiform body (8, 9). So, by following the blue fibres it is easy to demonstrate the rather complicated course of the sensory tract as it is thought to be at the present time. Thus it strikes the eye that in the medulla all fibres are situated on the side opposite to that which they have entered in the root. Laterally from the oblongata cross section the accessory nerve (33), which may be followed to its knee, juts out. Of other cranial nerves, the ascending part of the trigeminus (6) and of the glossopharyngeus (7) is shown: their fibres, dividing into fine brushes, end in the gelatinous columns, which in our model are represented in carved wood. Besides, the hypoglossal nerve (18) and part of the vagus roots (16, 17) are represented; from the nuclei of the latter the secondary (central) fibres are seen crossing over to the fillet. Of the connection between medulla and cerebellum only the spinal part of the restiform body could be shown, because the fibres descending from the cerebellum to the olivary body (10) only appear in more central parts of the medulla.

N.B.—As only part of the fibres have been embodied in our model, a few hanging wires had to be supported by metal props. These props, as all other parts of the model which serve merely technical purposes, have been painted in black, in order to be distinguished from the essential wires.

Both cross-sections bear handles which are to be used for turning the model, in order not to dirty the bright colours by touching.

The model is manufactured in the laboratory of Mr. R. Jung at Heidelberg. It is durable, and solidly as well as neatly made; with the exception of the nuclei and gelatinous substance, it is constructed entirely in metal. Care has been taken, in designing it, that new wires may easily be inserted, as well as other changes made.



Diagnosis of the Diseases which Clinically resemble Tabes Dorsalis (Pseudo-Tabes, Neuro-Tabes, &c.). By Dr. PAUL BLOCQ. *Gaz. des Hôpitaux*, March 22, 1890.

To simplify the differential diagnosis of the above affections a prelimary classification is adopted according to the presence of ataxy in

(1) Spinal affections; (2) diseases of the peripheral nerves; (3) neuroses.

In the following table the diseases in which tabetic symptoms are found are arranged in convenient order.

- *A.* Diseases in which tabetic (ataxic) symptoms occur.
  - 1. Spinal.
    - Locomotor ataxy.
    - Friedreich's hereditary ataxy.
    - Combined scleroses of the cord.
    - Syringomyelia.
  - 2. Peripheral nerves.
    - Pseudo-tabes.
      - *a.* Toxic.
        - Alcohol.
        - Arsenic.
        - Lead.
        - Diabetes.
      - *b.* Infectious: Beriberi.
    - "Neuro-Tabes."
  - 3. Neuroses.
    - Neurasthenic Pseudo-Tabes.
    - Hysterical Tabes.
- *B.* Diseases in which Tabetic (ataxic) symptoms are simulated.
  - Abasia.
  - Paramyoclonus Multiplex.
  - Thomsen's Disease.
  - Ataxy of Cerebellar type in
    - Cerebellar Tumours.
    - Menière's Disease.
    - Disseminated Sclerosis.

The author considers at length the diagnosis of these affections and gives a useful summary of the leading symptoms of each. With regard to the cases described by some authors under the title of pseudo-tabes, Dr. Blocq thinks that this name may now be abandoned, the cases really falling into two groups, the large majority being instances of paraplegia from peripheral neuritis, due to alcohol, lead, &c., the *paraplégies toxiques à type de flexion* of Professor Charcot, the others, at present comprising only a small number of cases, forming a provisional group, "*Ataxie périphérique*" *of Déjérine*, the exact nature of which must at present be regarded as doubtful.

J. MICHELL CLARKE.

Dana on the Cause of Chronic Degenerative Disease of the Spinal Cord.

In a paper read before the Congress of American Physicians and Surgeons (October, 1891), primary degenerations are classified as follows:—

1. Locomotor ataxia; 2. Lateral sclerosis; 3. Compound scleroses; 4. Multiple sclerosis; 5. Progressive muscular atrophy.

The recognized incurability of these affections renders the discovery of their cause a matter of great importance. The theory which Dr. Dana accepts as the most probable, is that which he terms "The Toxine Theory." Sclerosis is regarded not as a result of inflammation, a process which is now generally recognized as concerning only connective tissues and blood-vessels. The microscope indicates the chief change to be gradual decay and death of the nerve-fibre and cell. In some scleroses, as that of locomotor ataxia, this process is accompanied by the development of leucomaines or toxalbumins, which irritating substances may perhaps be charged with setting up those changes which are noticed in the connective tissue elements in this disease. Analogy certainly seems to favour the idea that degenerative processes in nerve-tissue are due to the presence of an organic poison of some sort which may be the result, either of a germ poison in the body, or of some change in the cells produced by a germ present at some previous time. The frequency with which degenerative changes follow typhoid fever, measles, scarlet fever, smallpox, and syphilis, is a circumstance which accords well with this theory.

Hock on Hæmatomyelia. (*The John Hopkins Hospital Reports*, August, 1891). Charcot and Hayem have doubted the existence of a primary hæmorrhage into the spinal cord unless produced by trauma, while Leyden, Eichorst, Gowers and Erb have admitted its existence, since it is often impossible, even with a microscopical examination, to determine whether we have to deal with a primary softening and a secondary hæmorrhage or with a primary hæmorrhage and a secondary softening, analogous to the condition in the brain. In the following cases trauma cannot be entirely excluded, still they cannot be classed under the head of the usual traumatic cases in which injury and concussion to the spine are immediately followed by a hæmorrhage, since in one case six days, in the other three weeks, intervened between the accident and the paralysis.

Case 1.—Patient was struck between the shoulders by a board falling from the height of eight to ten feet without producing any effects save some soreness next day, which soon passed off and left him quite well. Three weeks later he was suddenly seized with acute pain between the shoulders radiating down the spine and into the arms, and within *eight to ten*

minutes by complete sensory and motor paralysis from the arms down (with the exception of the higher-situated muscles of the arm). *Retentio urinæ et alvi.* Improvement in the left leg the following day. Comparatively rapid improvement also in the right leg and left arm, so that after three weeks there remained only paralysis of some of the muscles of the right arm, especially the forearm. On examination a month after, no sensory changes but paralysis with atrophy, showing the characteristic electrical changes of certain muscles of his right arm. The muscles concerned are the following, classing them under two heads: (1) showing complete loss of motion and reaction of degeneration; extensor ulnaris, extensor communis, all the flexors, small muscles of the hand; (2) showing decrease in electrical irritability and impairment of motion; lowest part of pectoralis major and triceps. Hence, following Thorburn's scheme, the lesion is on a level with the seventh and eighth cervical and first dorsal roots, and partly upward in the sixth cervical.

Case 2.—The patient fell from a train, sustained several injuries, but no perceptible one to the spine. No trace of paralysis for six days; at that time some pain in right extremities, and after some hours' sleep complete paralysis in the right leg and arm. On examination three months after, weakness of triceps, pectoralis, flexors and extensors, and small muscles of the right hand and arm, with decrease in electrical excitability in some of these muscles; also some visible atrophy; stiffness in the right leg; no atrophy or electrical changes in muscles of leg; loss of the sense of temperature and pain on the whole non-paralysed side. There is here then a case of Brown-Sequard's paralysis resembling the picture of a syringomyelia.

The author discusses, in the differential diagnosis:—1, Hæmaorachis or hæmorrhage into the spinal canal outside the cord; 2, Acute myelitis; 3, Acute ischæmic myelomalacia, and concludes that hæmatomyelia is the most tenable from the acute onset, the quick regression of some of the symptoms, and the electrical changes. The likelihood of an antecedent sub-acute myelitis before the hæmorrhage is also discussed, but not admitted. The delay in the effect of the concussion is certainly a most interesting feature, and while the presence or absence of myelitis may be doubtful, the sudden onset of the symptoms clearly points to hæmorrhage.

ERNEST WILLS.